6. Phase III: An Assessment for late-1990's Based on Microenvironmental Exposure Modeling

During the past decade, several changes have occurred that should alter the exposure of Californians to environmental tobacco smoke. Two of these changes are of particular importance: (a) the advent of AB13, which bans smoking in essentially all indoor workplaces; (b) a significant reduction in the prevalence of smoking among Californian adults. The purpose of this phase of the research is to estimate the distribution of exposures among Californian nonsmokers in the late 1990's to toxic air contaminants from environmental tobacco smoke, taking into account the effects of these changes.

6.1. Methods

6.1.1 Overview

The same approach was applied as in Phase II, with modifications and adjustments to the computational algorithm and the input data to account for the dominant changes in exposure conditions. Overall, a Monte-Carlo simulation technique was applied to generate probability distribution functions of 24-h exposures to TACs. Individual exposures were computed by combining activity pattern information with estimates of microenvironmental concentrations of TACs from ETS. The same four scenarios were used as in Phase II. Simulations were conducted separately for three age groups: adult and adolescent nonsmokers and children. As in Phase II, the base calculations were conducted for benzene; predictions for other species were determined by scaling the benzene results by the ratio of emission factors.

In the assessment, we assumed that AB13 was fully implemented and completely observed. Consequently, we assumed that no ETS exposure would occur in these microenvironments: occupational/office, retail/other, restaurant, bar/nightclub, and school. Exposures were modeled for the remaining microenvironments: residential, transportation, and residential guest. Activity pattern data from the APCR and CAP surveys were used without adjustment other than to account for changes in the likelihood of being exposed to ETS, as described below. Except for the changes noted below, the input variables needed to compute microenvironmental concentrations were those applied in Phase II (see Table 5.2).

6.1.2. Smoking Prevalence

Survey data show substantial decline in the prevalence of smoking among California adults during the past decade. These changes are expected to alter the likelihood of exposure to ETS as well as the concentrations to which nonsmokers are exposed.

We derived estimates of the percentage change in the prevalence of smoking between 1988 and 1998 and used these estimate to adjust ETS exposure estimates. The data we employed were from a behavioral survey of Californians (California Department of Health Services, 1998) which reported the annual prevalence of smoking among California adults beginning in 1984. The data for 1984-1997 are plotted in Figure 6.1 which demonstrates an overall downward trend in adult smoking prevalence between the mid to late 1980s and the mid to late 1990s. During the past several years, this trend has at least leveled and probably reversed. In fact, the survey data show a jump in smoking prevalence between 1995 and 1996 from 16.7% to 18.6%. In 1997, a statistically insignificant decline from 1996 was observed, from 18.6% to 18.2%.

It is not known to what extent the increase between 1995 and 1996 reflects a change in the definition of a smoker as opposed to a change in the trend observed for the previous decade of declining smoking prevalence. Prior to and including 1995, a person was classified as a smoker if he or she responded "yes" to the following two questions: (a) "have you smoked 100 cigarettes in your life?" and (b) "do you smoke now?" Beginning in 1996, the second question posed to determine whether or not a respondent smokes was changed to "do you smoke every day, some days, or not at all?" A person is now classified as a smoker if they indicate that they smoke either every day or some days.

To study the effect of changes in the definition of a smoker on prevalence, two groups of Californian adults (total sample size of 2000) were surveyed during the period July-December 1997 (Davis, 1998). One group was asked the earlier pair of questions; the other group was asked the other pair. The difference in smoking prevalence among these two groups was 1.1%, with the higher value corresponding to the 1996 and later set of questions.

In Figure 6.1, we plot for 1996-97 the smoking prevalence results based on the new pair of questions. We also plot estimated results for the old questions, obtained by subtracting 1.1%.

Overall, because of year-to-year fluctuations, because of changes in the definition of a smoker and because of the need to make predictions in advance of data availability, the prevalence of smoking among California adults for the late 1990s is uncertain. To capture this uncertainty in our model predictions, we have assigned to the simulation scenarios different smoking prevalence values for the late 1990s. The goal in selecting these values is to approximately bracket the true value without overestimating uncertainty.

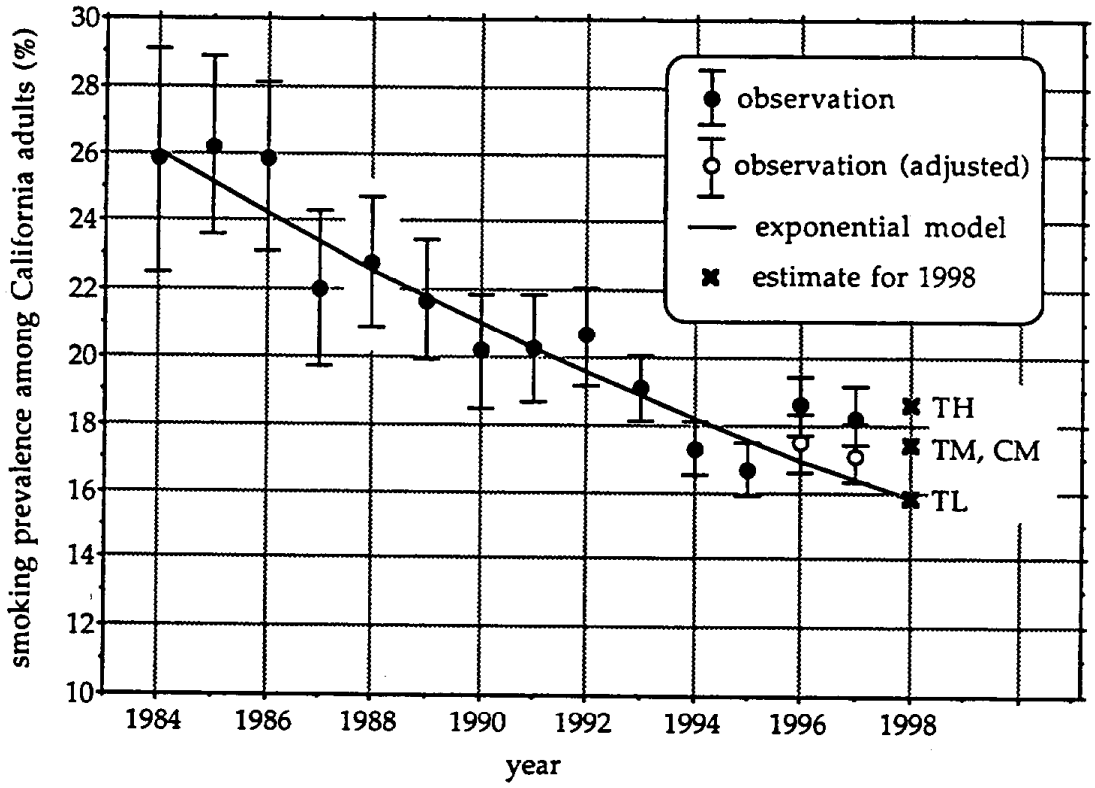

Figure 6.1 Prevalence of smoking among adult Californians, 1984-1997 (California Department of Health Services, 1998). The error bars indicate 95% confidence intervals. The exponential model fit is described in the text. The 1998 estimates are indicated for four simulation scenarios (TL - tracer low-range, TM - tracer mid-range, CM - completely mixed room model, and TH - tracer high-range).

Estimated values of smoking prevalence for the late 1990s are indicated by the three points plotted for 1998 in Figure 6.1. For the TH scenario, we selected the highest recent-year smoking prevalence (1996, unadjusted): 18.6%. The predicted percentage reduction in smoking prevalence for this scenario between the late 1980s (for 1988 the prevalence was 22.8%) and the late 1990s (1998) is 18%.[1] For the TM and CM scenarios, we selected the average adjusted smoking prevalence for 1996 and 1997: 17.4%. For this scenario, the predicted percentage reduction in smoking prevalence between Phases II and III is 24%.[2]

For the TL scenario, we applied a regression model to the 1984-97 prevalence data (using the adjusted data for 1996 and 1997). We used an exponential decay model which assumes that the fractional change in smoking prevalence is constant from one year to the next. The regression was done using a weighted least-squares procedure (Bevington and Robinson, 1992) with weights inversely proportional to size of the error bars. The fitted regression, plotted in Figure 6.1, is described by this equation:

$$P_y = 26.03\% \times \exp[-0.0357\ (y-1984)] \qquad (6.1)$$

where P_y is the prevalence of adult smoking in year y. According to this model, the change in smoking prevalence from 1988 to 1998 would be from 22.6% to 15.8%, a reduction of 30%.[3]

There is no fundamental basis on which to select one estimate over another. None is derived from first principles; rather they are based on empirical or statistical descriptions of complex psychosocial behaviors. Smoking prevalence is significantly influenced by tobacco taxes, antismoking publicity campaigns, cigarette advertising, and demographic shifts. The model represented by equation 6.1, for example, is a convenient estimation tool for making a short-term extrapolation based on past trends and should not be viewed as a robust predictor of future behavior.

We note that the value for smoking prevalence from survey data for 1988, 22.8%, corresponds well with the finding from the Activity Patterns of California Residents, which revealed a smoking prevalence among adults of 22.5% (Jenkins et al., 1992).

Changes in smoking prevalence can affect ETS exposures in two ways. The frequency of exposures might change. Also the concentrations of ETS constituents in environments where smoking occurs may change. With AB13 in effect, remaining environments in which ETS exposures can occur will typically have small numbers of smokers. Therefore, we expect that the

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[1] 18% = (22.8% - 18.6%)/22.8% × 100%.

[2] 24% = (22.8%-17.4%)/22.8% × 100%.

[3] 30% = (22.6%-15.8%)/22.6% × 100%.

dominant influence of changes in smoking prevalence will be on the frequency of exposures, rather than on their intensity.

6.1.2.1 Residential Microenvironment

For adults, we assumed that the likelihood that any individual be exposed to ETS in their own residence was reduced by the same percentage as the fractional reduction in smoking prevalence, denoted R (0.18 for scenario TH, 0.24 for scenarios TM and CM, and 0.30 for scenario TH). This assumption follows from the treatment in the Phase II CM scenario in which we assumed only one smoking resident was present in an exposed adult's household. In the Phase III simulations, this likelihood was applied stochastically. For each individual that was exposed to ETS in their own residence in 1988, a random number was selected and used to predict whether that person would be exposed in 1998. For each individual, the likelihood was (1-R) × 100% that they were still exposed and, if so, their exposure was computed as in Phase II (except for a minor correction for cigarette consumption rate, as described in §6.1.3). In the other outcome, which occurred with probability R × 100% , the residential exposure was assumed to be zero.

For all population age groups, the same approach was used for the three tracer scenarios (TL, TM, and TH). However, for children and adolescents in the CM scenario, the approach was modified somewhat to be consistent with our treatment of multiple smokers in that setting in Phase II.

Recall that in the Phase II CM simulation, an adolescent or child who was exposed to ETS in their residence had a 72% chance of being exposed to smoke from a single smoker, a 26% chance of being exposed to the smoke from two smokers, and a 2% chance of being exposed to the smoke from three smokers. We modeled the effect of changes in smoking prevalence on these probabilities by assuming that the likelihood that a smoker in 1988 was a smoker in 1998 was the same for all smokers. Lacking detailed data on how the numbers of multiple smoker households have changed with time, we judged this approach to be the best approximation of reality we could achieve. With this assumption, by adding conditional probabilities, we determined that the following probability would apply to an adolescent or child exposed at home in 1988:

$$\text{chance of being unexposed in 1998, given that exposure occurred in 1988} = R \times 72\% + R^2 \times 26\% + R^3 \times 2\% \qquad (6.2)$$

Applying this equation for scenario TH (R = 0.18), the likelihood of becoming unexposed is 14% and therefore the likelihood of remaining exposed is 86%. Likewise, the probability of becoming unexposed is 19% for scenario TM and CM and 24% for scenario TL. Among those who remain exposed, the number of smokers in the household is also determined by applying conditional probabilities.

The likelihood of exposure to one smoker is

$$\text{probability of exposure to one smoker in 1998, given that exposure occurs in 1998} = \frac{(1\text{-}R)\times 72\% + 2\times(1\text{-}R)\times R\times 26\% + 3\times(1\text{-}R)\times R^2\times 2\%}{100\% - \left[R\times 72\% + R^2\times 26\% + R^3\times 2\%\right]} \quad (6.3)$$

For two smokers, the corresponding expression is

$$\text{probability of exposure to two smokers in 1998, given that exposure occurs in 1998} = \frac{(1\text{-}R)^2 \times 26\% + 3 \times (1\text{-}R)^2 \times R \times 2\%}{100\% - \left[R \times 72\% + R^2 \times 26\% + R^3 \times 2\%\right]} \quad (6.4)$$

And, for three smokers, the expression is

$$\text{probability of exposure to three smokers in 1998, given that exposure occurs in 1998} = \frac{(1\text{-}R)^3 \times 2\%}{100\% - \left[R \times 72\% + R^2 \times 26\% + R^3 \times 2\%\right]} \quad (6.5)$$

The results from these expressions are as follows. For scenario TH (R = 0.18), the probabilities of 1, 2, or 3 smokers in a household (given that there is at least one) are 77.6%, 21.1%, and 1.3%, respectively. For scenarios TM and CM (R = 0.24), the corresponding probabilities are 79.4%, 19.5%, and 1.1%. And for scenario TL (R = 0.30), the probabilities are 81.2%, 17.9%, and 0.9%.

During the Monte-Carlo simulations, for each adolescent or child who was exposed at home in 1988 we first selected a random number to decide whether or not they were exposed at home in 1998. If so, we modeled their exposure as in the Phase II CM scenario, but with altered probabilities of multiple smokers. In addition to altering the probability of exposure, the smoking rate was adjusted to account for changes in cigarette consumption.

6.1.2.2 Transportation Microenvironment

In Phase II, the transportation microenvironment was modeled assuming that only one occupant smoked cigarettes during a given trip. Following that treatment, for Phase III, we assumed that the proportion of individuals exposed to ETS would be reduced in direct proportion to the change in smoking prevalence. A probabilistic selection was applied for each individual who was exposed in the transportation microenvironment in 1988 such that the likelihood of being exposed in 1998 was 100%-R. This approach underestimates exposure in circumstances where more than one individual smokes during a trip. Data are lacking to meaningfully relax this assumption. In our judgment, bias in the overall results associated by assuming a single smoker in a vehicle is not likely to be large. Concentrations were simulated as in Phase II, with an adjustment for cigarette smoking rate as described below.

6.1.2.3 Residential Guest

As with the transportation microenvironment, we assumed that the proportion of exposed individuals would be reduced in direct proportion to the change in smoking prevalence. Again, this treatment follows from the assumption that most exposures would result from the presence of a single smoker. This approximation represents a practical necessity as data on the occurrence of multiple smokers in "residential guest" settings are lacking. The residential guest scenarios were all based on the tracer method of calculating microenvironmental concentrations. The microenvironmental concentrations were adjusted to account for changes in cigarette consumption rates.

6.1.3. Cigarette Consumption

Concentrations of ETS constituents in microenvironments could be altered by any shift in cigarette consumption patterns. The previous section described how changes in smoking prevalence were incorporated into the model predictions. The effects of changes in daily cigarette consumption per smoker are addressed here.

Evidence indicates that changes in cigarette consumption rates are relatively small between the late 1980s and late 1990s. Figure 6.2 shows annual survey results for the number of cigarettes smoked per day by Californian adult smokers (Davis, 1998). For Phase III modeling, we used these data as a basis for selecting different values for cigarette consumption rate in the late 1990's for different simulation scenarios. As with smoking prevalence, the goal was to bracket the true consumption rate for 1998 without overestimating the uncertainty. For the TH and TL scenarios, we respectively used the maximum and minimum recent-year cigarette consumption rate: 16.9 cig/d (1996) and 14.6 cig/d (1995). For the TM and CM scenarios, we used the arithmetic mean of the last three years of results (1995-1997): 16.1 cig/d. The 1988 survey result, 16.2 cig/d, was assumed to apply for the late 1980's. Thus, the percentage change from the late 1980s to the late 1990s in cigarette consumption rate is estimated to be +4.3% for scenario TH, -0.7% for scenarios TM and CM, and -9.9% for scenario TL.

The effects of the change in cigarette consumption were modeled differently for the CM and tracer scenarios. In the CM scenarios, we adjusted the cigarette smoking rate relative to the 1988 value according to the estimates just described. For the adult and adolescent populations, the effect of this change was modeled using the same matchmaking scheme as in Phase II, but adjusting by a fixed percentage of that individual's at-home cigarette consumption. For children, a different approach was used, as in Phase II. Here, the rate of at-home cigarette consumption per smoker was selected from a lognormal distribution. The GM was adjusted upward or downward from the Phase II estimate (0.31 cigarettes $smoker^{-1}$ h^{-1}, see §5.1.3.1.1) according to the estimated percentage change. The GSD was unchanged (3.0).

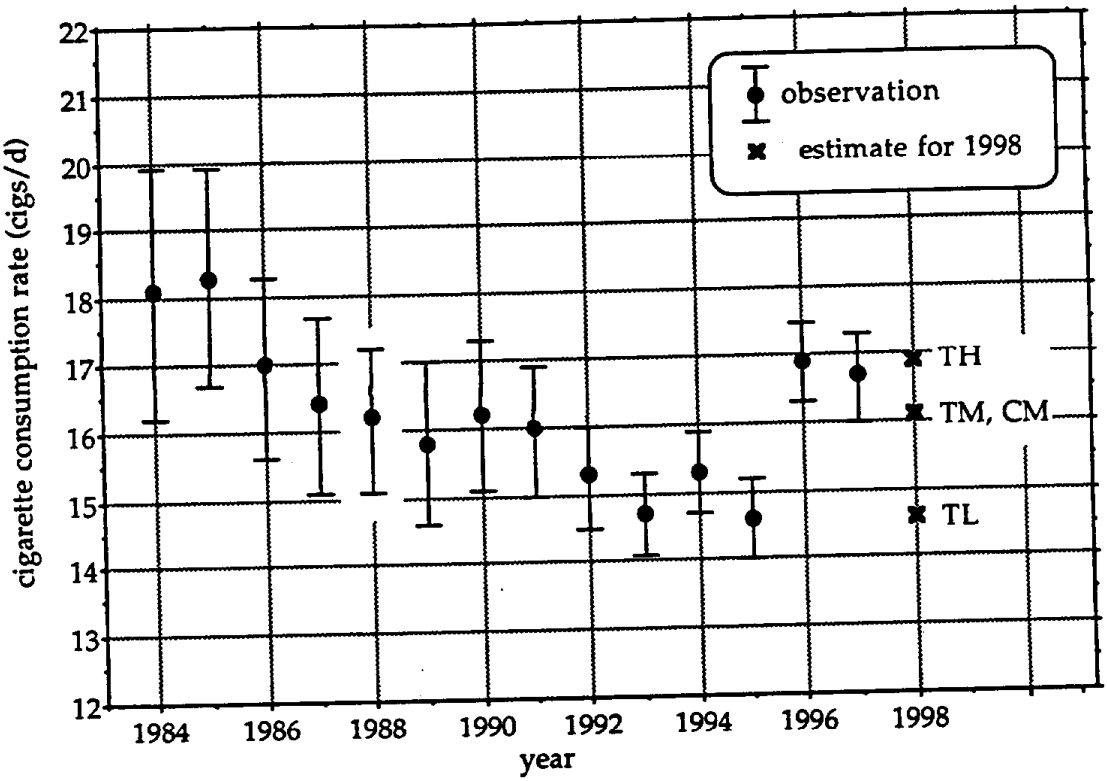

Figure 6.2 Averaged daily consumption of cigarettes by Californian smokers, 1984-1997 (California Department of Health Services, 1998). The error bars indicate 95% confidence intervals. The 1998 estimates are indicated for four simulation scenarios (TL - tracer low-range, TM - tracer mid-range, CM - completely mixed room model, and TH - tracer high-range).

For the tracer scenarios, we assumed that the different cigarette consumption rate would produce proportionately different microenvironmental concentrations. Thus, for Phase III, we selected concentrations from the same lognormal distributions as in Phase II, except that the GM was adjusted by the estimated percentage change in cigarette consumption rate (the GSD was unchanged).

6.2 Results and Discussion

6.2.1 Proportion of Nonsmokers Exposed

Table 6.1 shows the total proportion of nonsmokers predicted to be exposed to ETS on any day in the simulated microenvironments. For adults, the percentage drops from 52% in the late 1980's to 16-19% (depending on scenario) in the late 1990's. For adolescents the change is from 62% to 33-35% and for children the change is from 33% to 21-23%. The improvement is much larger for adults because many of them were only exposed in the late 1980's in microenvironments in which smoking is no longer permitted. For children, especially, most of their exposure to ETS occurs in microenvironments in which smoking is not regulated. The predicted proportion of adults exposed has declined both because of a reduction in the prevalence of smoking and also because of restrictions prohibiting smoking in many indoor environments. For children, the dominant effect is only due to smoking prevalence changes.

Figure 6.3 shows the proportion of nonsmokers predicted to be exposed in the microenvironments where exposure can still (legally) occur. As shown in the figure, 5-9% of nonsmoking Californian adults are predicted to be exposed to ETS on a given day in each of three settings: their own residence (8-9%), in transportation microenvironments (6-7%), and as a residential guest (5-7%). The corresponding percentages for adolescents are consistently higher: 18-19% in their own residence, 12-14% in transportation microenvironments, and 13-19% as a guest in a residence. Exposure prevalences for children are predicted to be intermediate between adults and adolescents. On a given day, 14-17% of California children are predicted to be exposed to ETS in their homes, down from 20% exposed in the late 1980's. The proportions of children exposed to ETS in a transportation microenvironment or as a residential guest are approximately 8% and 5%, respectively.

Table 6.1 Proportion of nonsmoking California population predicted to be exposed to ETS on a given day in one or more simulated microenvironments [1,2]

	adults	adolescents	children
Phase II — late 1980's			
all scenarios	52%	63%	33%
Phase III — late 1990's			
scenario TL	16%	33%	21%
scenario TM	18%	35%	22%
scenario CM	18%	35%	23%
scenario TH	19%	35%	23%

[1] See also Figure 3.4.

[2] Scenarios: TL - tracer low-range, TM - tracer mid-range, CM - completely mixed room model, TH - tracer high-range.

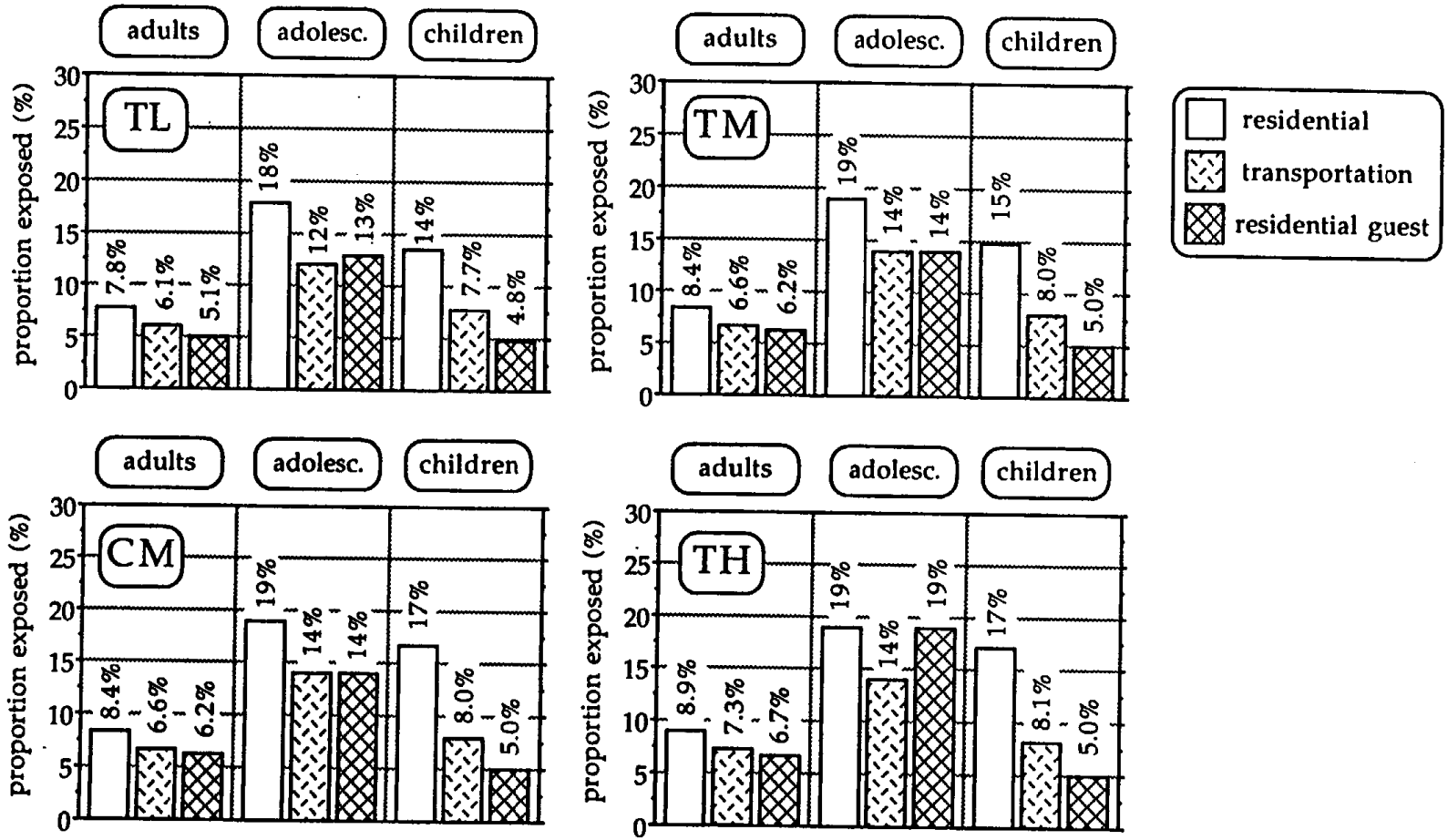

Figure 6.3 Percentage (weighted) of nonsmokers predicted to be exposed to ETS in different microenvironments in late 1990's. Predictions differ among scenarios mainly because of different estimates of the prevalence of smoking in 1998. (Scenarios: TL - tracer low-range; TM - tracer mid-range; CM - completely mixed room model; TH - tracer high-range.)

The data presented in Figure 6.3 are based on the activity pattern survey data (Wiley et al., 1991a, 1991b), with modeling adjustments to account for the lower prevalence of smoking in the late 1990's. For each individual who reported being exposed in the late 1980's, and in each microenvironment in which that person reported exposure, we stochastically modeled whether exposure would be expected in the late 1990's. For all cases other than the residential setting according to the CM scenario for adolescents and children, the probability of being exposed in the late 1990's was taken as the ratio of adult smoking prevalence in the late 1990's to the late 1980's. The proportion exposed in each microenvironment in the late 1990's would be, therefore, approximately (1-R) × 100% of the proportion exposed in the late 1980's. (The results do not match exactly because the probability was applied stochastically for each exposed individual.)

6.2.2 Predicted Toxic Air Contaminant Exposure from ETS

6.2.2.1 Total Exposure

Tables 6.2-6.4 present summary statistics of the probability distribution functions for exposure to all toxic air contaminants studied, as predicted for the late 1990's. These tables are constructed in the same style as Tables 5.3-5.5 for the late 1980's. As before, the statistics apply only to those nonsmokers exposed to ETS on a given day. Figure 6.4 displays similar information in a graphical format, showing for each population group and scenario the arithmetic mean plus selected percentiles for the distributions of exposure to benzene from ETS. For those exposed, the average contribution of ETS to benzene exposure is predicted to be in the range 12-21 $\mu g\ h\ m^{-3}$ for adults, 9-22 $\mu g\ h\ m^{-3}$ for adolescents, and 12-28 $\mu g\ h\ m^{-3}$ for children. The corresponding ranges for the 90th percentiles of each distribution are 29-53 $\mu g\ h\ m^{-3}$ (for adults), 21-46 $\mu g\ h\ m^{-3}$ (for adolescents), and 28-61 $\mu g\ h\ m^{-3}$ (for children).

The predicted cumulative distributions of exposure to benzene from ETS for the exposed nonsmoking Californian population are shown in Figure 6.5, again segregated by age group and scenario. Figure 5.2 showed analogous results for Phase II. As in that case, the distributions conform approximately to lognormality, but again exhibit a bowing downward such that the best-fit lognormal distribution tends to overpredict the high-percentile concentrations and underpredict the median.

Table 6.2. Statistical parameters for total daily exposure of the California adult nonsmoking population to toxic air contaminants from environmental tobacco smoke, predicted for late 1990's. [1, 2]

species/ scenario	*AM* ($\mu g\ h\ m^{-3}$)	*SD* ($\mu g\ h\ m^{-3}$)	*GM* ($\mu g\ h\ m^{-3}$)	*GSD*
acetaldehyde				
TL	64	106	27	4.9
TM	79	120	31	5.1
CM	69	110	28	4.9
TH	110	140	49	4.9
acetonitrile [3]				
TL	49	82	21	4.9
TM	61	94	24	5.1
CM	53	86	21	4.9
TH	86	106	38	4.9
acrylonitrile				
TL	2.9	4.9	1.2	4.9
TM	3.7	5.6	1.4	5.1
CM	3.2	5.1	1.3	4.9
TH	5.1	6.3	2.2	4.9
benzene				
TL	12	20	5.1	4.9
TM	15	23	5.9	5.1
CM	13	21	5.2	4.9
TH	21	26	9.2	4.9
1,3-butadiene				
TL	4.5	7.5	1.9	4.9
TM	5.6	8.6	2.2	5.1
CM	4.9	7.9	1.9	4.9
TH	7.9	9.7	3.4	4.9
2-butanone				
TL	8.6	14	3.7	4.9
TM	10.8	16	4.2	5.1
CM	9.3	15	3.7	4.9
TH	15	19	6.6	4.9
o-cresol				
TL	1.03	1.7	0.44	4.9
TM	1.3	2.0	0.51	5.1
CM	1.1	1.8	0.45	4.9
TH	1.8	2.2	0.79	4.9
m,p-cresol				
TL	2.5	4.1	1.04	4.9
TM	3.1	4.7	1.2	5.1
CM	2.7	4.3	1.06	4.9
TH	4.3	5.3	1.9	4.9

[1] AM–arithmetic mean, SD–arithmetic standard deviation, GM–geometric mean, GSD–geometric standard deviation; TL - tracer low exposure, TM - tracer mid-range exposure, CM - completely mixed room model, TH - tracer high exposure.

[2] Results apply to the proportion of the adult nonsmoking population in California predicted to be exposed during a day to ETS (TL - 16%; TM - 18%; CM - 18%; TH - 19%).

[3] Parameters estimated based on the ratio of emission factors for acetonitrile (1145 $\mu g\ cig^{-1}$) to benzene (280 $\mu g\ cig^{-1}$) reported by Martin et al., 1997; acetonitrile emissions not measured by Daisey et al.

Table 6.2. (continued)

species/ scenario	*AM* ($\mu g\ h\ m^{-3}$)	*SD* ($\mu g\ h\ m^{-3}$)	*GM* ($\mu g\ h\ m^{-3}$)	*GSD*
ethyl acrylate				
TL	< 0.09	—	< 0.04	—
TM	< 0.11	—	< 0.04	—
CM	< 0.10	—	< 0.04	—
TH	< 0.16	—	< 0.07	—
ethylbenzene				
TL	3.8	6.4	1.6	4.9
TM	4.8	7.4	1.9	5.1
CM	4.2	6.7	1.7	4.9
TH	6.7	8.3	2.9	4.9
formaldehyde				
TL	39	65	16	4.9
TM	48	74	19	5.1
CM	42	68	17	4.9
TH	68	84	30	4.9
n-nitrosodimethylamine				
TL	0.017	0.028	0.007	4.9
TM	0.021	0.032	0.008	5.1
CM	0.018	0.029	0.007	4.9
TH	0.029	0.037	0.013	4.9
phenol				
TL	8.3	14	3.5	4.9
TM	10.4	16	4.1	5.1
CM	9.0	15	3.6	4.9
TH	15	18	6.4	4.9
styrene				
TL	4.3	7.2	1.8	4.9
TM	5.4	8.3	2.1	5.1
CM	4.7	7.6	1.9	4.9
TH	7.6	9.4	3.3	4.9
toluene				
TL	19	32	8.2	4.9
TM	24	37	9.5	5.1
CM	21	34	8.4	4.9
TH	34	42	15	4.9
o-xylene				
TL	2.0	3.3	0.84	4.9
TM	2.5	3.8	0.97	5.1
CM	2.1	3.5	0.86	4.9
TH	3.5	4.3	1.5	4.9
m,p-xylene				
TL	8.8	15	3.8	4.9
TM	11	17	4.3	5.1
CM	9.6	15	3.8	4.9
TH	15	19	6.8	4.9

Table 6.3. Statistical parameters for daily exposure of Californian adolescent nonsmokers to toxic air contaminants from environmental tobacco smoke, predicted for late 1990's. [1,2]

species/ scenario	*AM* ($\mu g\ h\ m^{-3}$)	*SD* ($\mu g\ h\ m^{-3}$)	*GM* ($\mu g\ h\ m^{-3}$)	*GSD*
acetaldehyde				
TL	48	85	21	4.3
TM	74	101	32	4.8
CM	64	90	26	4.6
TH	120	130	58	4.4
acetonitrile [3]				
TL	37	65	16	4.3
TM	57	78	25	4.8
CM	49	70	20	4.6
TH	90	102	45	4.4
acrylonitrile				
TL	2.2	3.9	1.0	4.3
TM	3.4	4.6	1.5	4.8
CM	2.9	4.1	1.2	4.6
TH	5.4	6.1	2.7	4.4
benzene				
TL	9.1	16	4.0	4.3
TM	14	19	6.1	4.8
CM	12	17	5.0	4.6
TH	22	25	11	4.4
1,3-butadiene				
TL	3.4	6.0	1.5	4.3
TM	5.2	7.1	2.3	4.8
CM	4.5	6.4	1.9	4.6
TH	8.2	9.4	4.1	4.4
2-butanone				
TL	6.5	11	2.9	4.3
TM	10.0	14	4.4	4.8
CM	8.6	12	3.6	4.6
TH	16	18	7.9	4.4
o-cresol				
TL	0.78	1.4	0.34	4.3
TM	1.2	1.6	0.53	4.8
CM	1.03	1.5	0.43	4.6
TH	1.9	2.2	0.95	4.4
m,p-cresol				
TL	1.9	3.3	0.82	4.3
TM	2.9	3.9	1.2	4.8
CM	2.5	3.5	1.02	4.6
TH	4.5	5.1	2.2	4.4

[1] AM–arithmetic mean, SD–arithmetic standard deviation, GM–geometric mean, GSD–geometric standard deviation; TL - tracer low exposure, TM - tracer mid-range exposure, CM - completely mixed room model, TH - tracer high exposure.

[2] Results apply to the proportion of the adolescent nonsmoking population in California that is predicted to experience some exposure during a day to ETS (TL - 33%; TM - 35%; CM - 35%; TH - 35%).

[3] Parameters estimated based on the ratio of emission factors for acetonitrile (1145 $\mu g\ cig^{-1}$) to benzene (280 $\mu g\ cig^{-1}$) reported by Martin et al., 1997; acetonitrile emissions not measured by Daisey et al.

Table 6.3. (continued)

species/ scenario	*AM* ($\mu g\ h\ m^{-3}$)	*SD* ($\mu g\ h\ m^{-3}$)	*GM* ($\mu g\ h\ m^{-3}$)	*GSD*
ethyl acrylate				
TL	< 0.07	—	< 0.03	—
TM	< 0.10	—	< 0.05	—
CM	< 0.09	—	< 0.04	—
TH	< 0.16	—	< 0.08	—
ethylbenzene				
TL	2.9	5.1	1.3	4.3
TM	4.5	6.1	2.0	4.8
CM	3.8	5.4	1.6	4.6
TH	7.0	8.0	3.5	4.4
formaldehyde				
TL	29	52	13	4.3
TM	45	61	20	4.8
CM	39	55	16	4.6
TH	71	81	35	4.4
n-nitrosodimethylamine				
TL	0.013	0.022	0.006	4.3
TM	0.020	0.027	0.009	4.8
CM	0.017	0.024	0.007	4.6
TH	0.031	0.035	0.015	4.4
phenol				
TL	6.3	11	2.8	4.3
TM	9.7	13	4.2	4.8
CM	8.3	12	3.5	4.6
TH	15	17	7.6	4.4
styrene				
TL	3.3	5.8	1.4	4.3
TM	5.1	6.9	2.2	4.8
CM	4.3	6.2	1.8	4.6
TH	8.0	9.1	4.0	4.4
toluene				
TL	15	26	6.5	4.3
TM	23	31	9.9	4.8
CM	19	27	8.1	4.6
TH	36	40	18	4.4
o-xylene				
TL	1.5	2.6	0.66	4.3
TM	2.3	3.1	1.01	4.8
CM	2.0	2.8	0.83	4.6
TH	3.6	4.1	1.8	4.4
m,p-xylene				
TL	6.7	12	2.9	4.3
TM	10.3	14	4.5	4.8
CM	8.8	13	3.7	4.6
TH	16	18	8.1	4.4

Table 6.4. Statistical parameters for total daily exposure of the California children population to toxic air contaminants from environmental tobacco smoke, predicted for late 1990's. [1,2]

species/ scenario	*AM* ($\mu g\ h\ m^{-3}$)	*SD* ($\mu g\ h\ m^{-3}$)	*GM* ($\mu g\ h\ m^{-3}$)	*GSD*
acetaldehyde				
TL	64	95	31	4.2
TM	95	130	45	4.6
CM	101	140	49	4.3
TH	150	150	79	3.9
acetonitrile [3]				
TL	49	74	24	4.2
TM	74	98	35	4.6
CM	78	110	38	4.3
TH	110	110	61	3.9
acrylonitrile				
TL	2.9	4.4	1.4	4.2
TM	4.4	5.9	2.1	4.6
CM	4.6	6.6	2.2	4.3
TH	6.8	6.8	3.7	3.9
benzene				
TL	12	18	5.8	4.2
TM	18	24	8.5	4.6
CM	19	27	9.2	4.3
TH	28	28	15	3.9
1,3-butadiene				
TL	4.5	6.7	2.2	4.2
TM	6.7	9.0	3.2	4.6
CM	7.1	10.1	3.4	4.3
TH	10.5	10.5	5.6	3.9
2-butanone				
TL	8.6	13	4.2	4.2
TM	13	17	6.1	4.6
CM	14	19	6.6	4.3
TH	20	20	10.8	3.9
o-cresol				
TL	1.03	1.6	0.50	4.2
TM	1.6	2.1	0.73	4.6
CM	1.6	2.3	0.79	4.3
TH	2.4	2.4	1.3	3.9
m,p-cresol				
TL	2.5	3.7	1.2	4.2
TM	3.7	4.9	1.7	4.6
CM	3.9	5.5	1.9	4.3
TH	5.7	5.7	3.1	3.9

[1] AM–arithmetic mean, SD–arithmetic standard deviation, GM–geometric mean, GSD–geometric standard deviation; TL - tracer low exposure, TM - tracer mid-range exposure, CM - completely mixed room model, TH - tracer high exposure.

[2] Results apply to the proportion of children in California for whom some exposure to ETS is predicted to occur during a day (TL - 21%; TM - 22%; CM - 23%; TH - 23%).

[3] Parameters estimated based on the ratio of emission factors for acetonitrile (1145 $\mu g\ cig^{-1}$) to benzene (280 $\mu g\ cig^{-1}$) reported by Martin et al., 1997; acetonitrile emissions not measured by Daisey et al.

Table 6.4. (continued)

species/ scenario	AM ($\mu g\ h\ m^{-3}$)	SD ($\mu g\ h\ m^{-3}$)	GM ($\mu g\ h\ m^{-3}$)	GSD
ethyl acrylate				
TL	< 0.09	—	< 0.04	—
TM	< 0.13	—	< 0.06	—
CM	< 0.14	—	< 0.07	—
TH	< 0.21	—	< 0.11	—
ethylbenzene				
TL	3.8	5.8	1.9	4.2
TM	5.8	7.7	2.7	4.6
CM	6.1	8.6	2.9	4.3
TH	9.0	9.0	4.8	3.9
formaldehyde				
TL	39	58	19	4.2
TM	58	77	27	4.6
CM	61	87	30	4.3
TH	90	90	48	3.9
n-nitrosodimethylamine				
TL	0.017	0.025	0.008	4.2
TM	0.025	0.034	0.012	4.6
CM	0.027	0.038	0.013	4.3
TH	0.039	0.039	0.021	3.9
phenol				
TL	8.3	12	4.0	4.2
TM	12	17	5.9	4.6
CM	13	19	6.4	4.3
TH	19	19	10.4	3.9
styrene				
TL	4.3	6.5	2.1	4.2
TM	6.5	8.7	3.1	4.6
CM	6.9	9.8	3.3	4.3
TH	10.1	10.1	5.4	3.9
toluene				
TL	19	29	9.4	4.2
TM	29	39	14	4.6
CM	31	44	15	4.3
TH	45	45	24	3.9
o-xylene				
TL	2.0	3.0	0.96	4.2
TM	3.0	4.0	1.4	4.6
CM	3.1	4.5	1.5	4.3
TH	4.6	4.6	2.5	3.9
m,p-xylene				
TL	8.8	13	4.3	4.2
TM	13	18	6.3	4.6
CM	14	20	6.8	4.3
TH	21	21	11	3.9

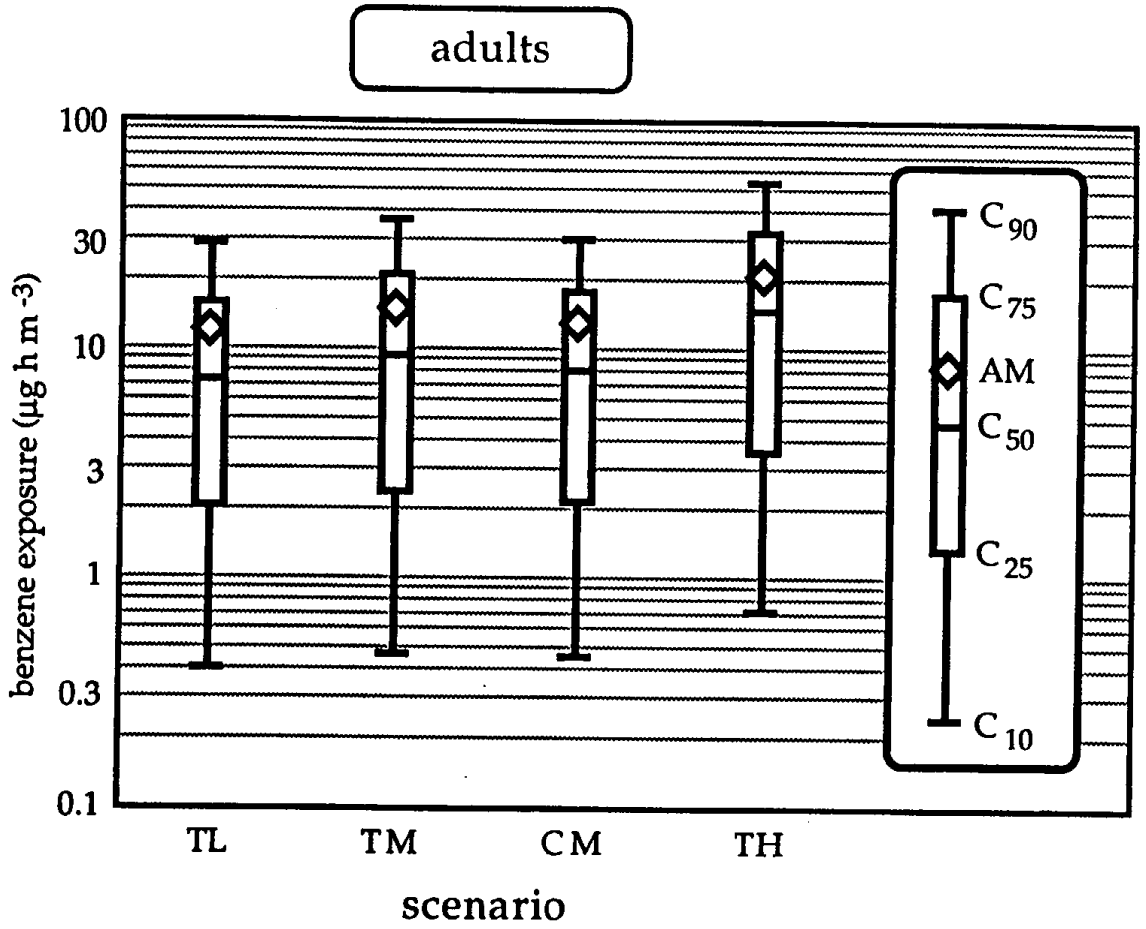

Figure 6.4a A whisker diagram summarizing distributional results for total exposure to benzene from ETS for nonsmokers in all four scenarios, predicted for late 1990's for nonsmoking Californian adults. (Scenarios: TL - tracer low-range, TM - tracer mid-range; CM - completely mixed room model; TH - tracer high-range. AM is the arithmetic mean and C_i represents the i^{th} percentile of the distribution.)

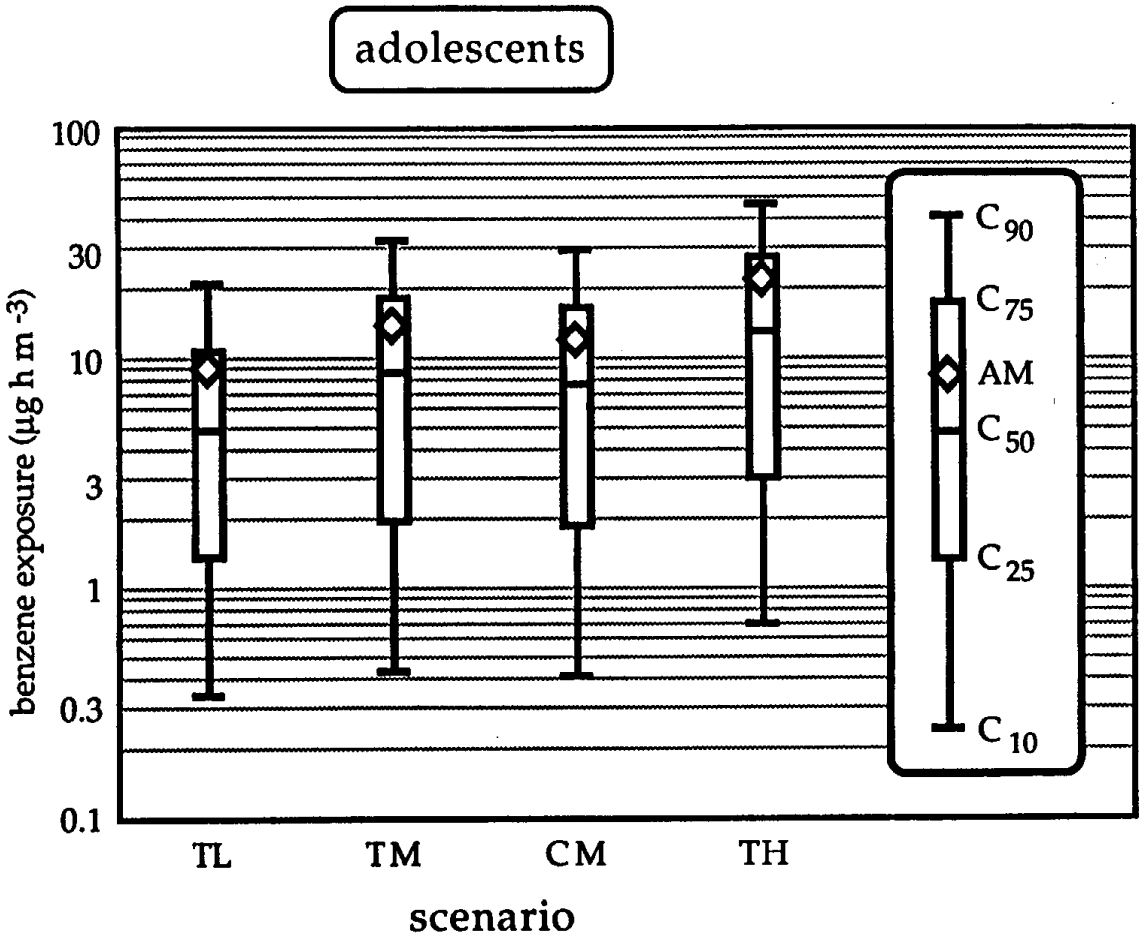

Figure 6.4b A whisker diagram summarizing distributional results for total exposure to benzene from ETS for nonsmokers in all four scenarios, predicted for late 1990's for nonsmoking Californian adolescents. (Scenarios: TL - tracer low-range, TM - tracer mid-range; CM - completely mixed room model; TH - tracer high-range. AM is the arithmetic mean and C_i represents the i^{th} percentile of the distribution.)

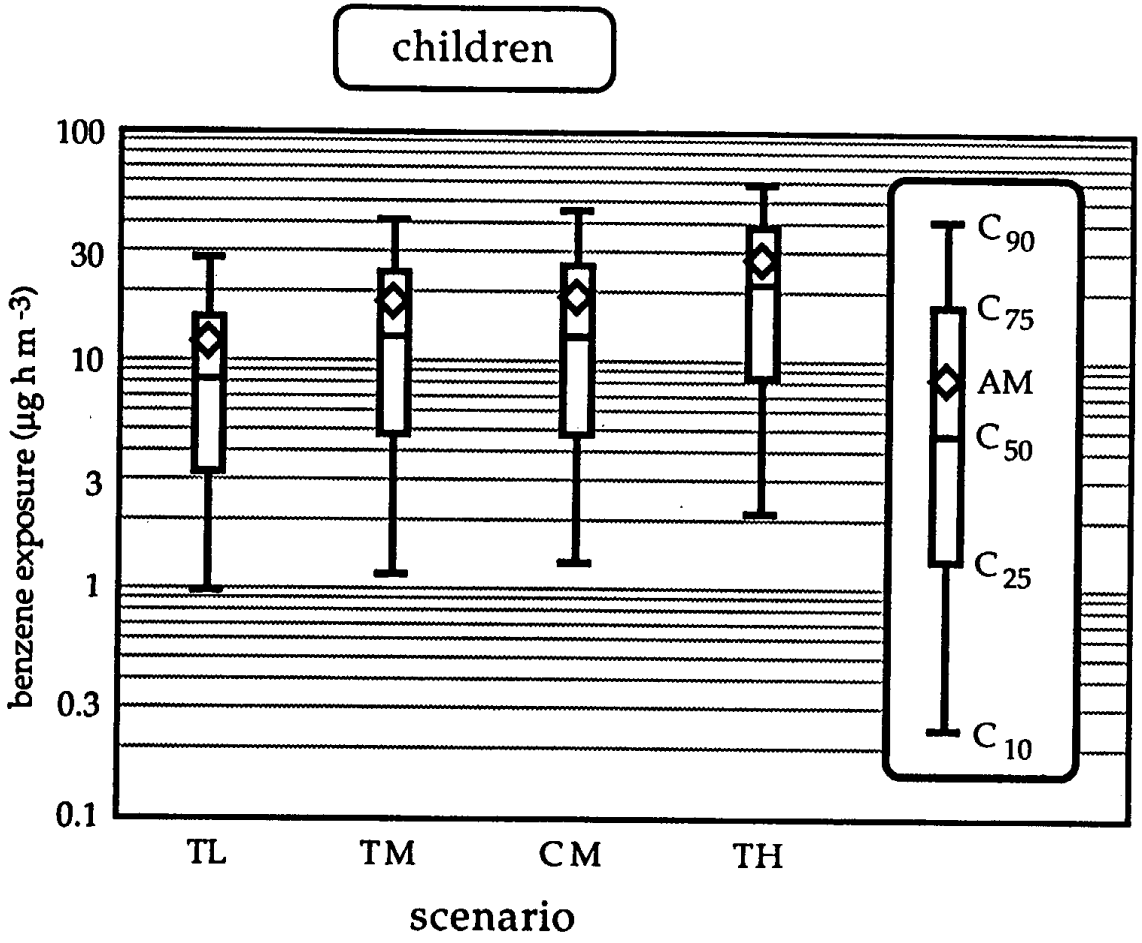

Figure 6.4c A whisker diagram summarizing distributional results for total exposure to benzene from ETS for nonsmokers in all four scenarios, predicted for late 1990's for Californian children. (Scenarios: TL - tracer low-range, TM - tracer mid-range; CM - completely mixed room model; TH - tracer high-range. AM is the arithmetic mean and C_i represents the i^{th} percentile of the distribution.)

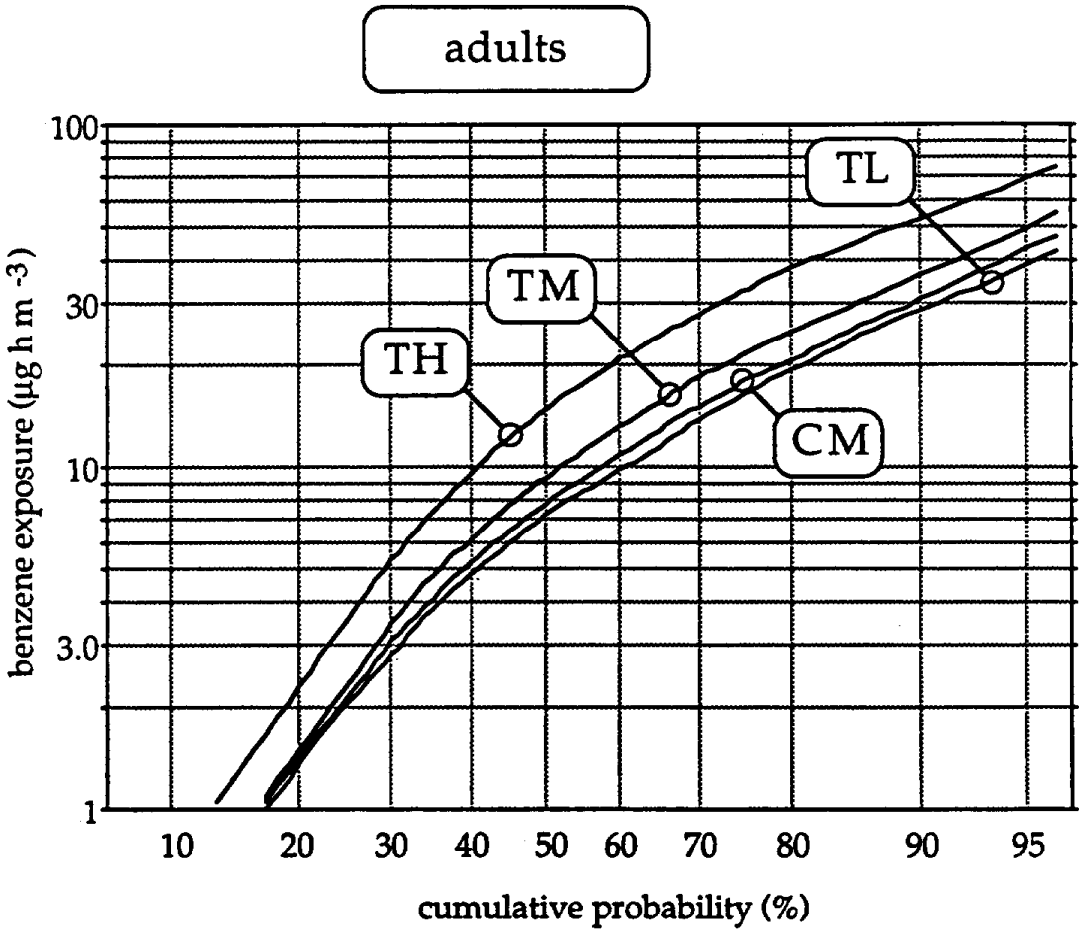

Figure 6.5a Lognormal-probability plot of the distributions of total exposure to benzene from ETS, separated by scenario, predicted for late 1990's for nonsmoking Californian adults. (Scenarios: TL - tracer low-range, TM - tracer mid-range; CM - completely mixed room model; TH - tracer high-range.)

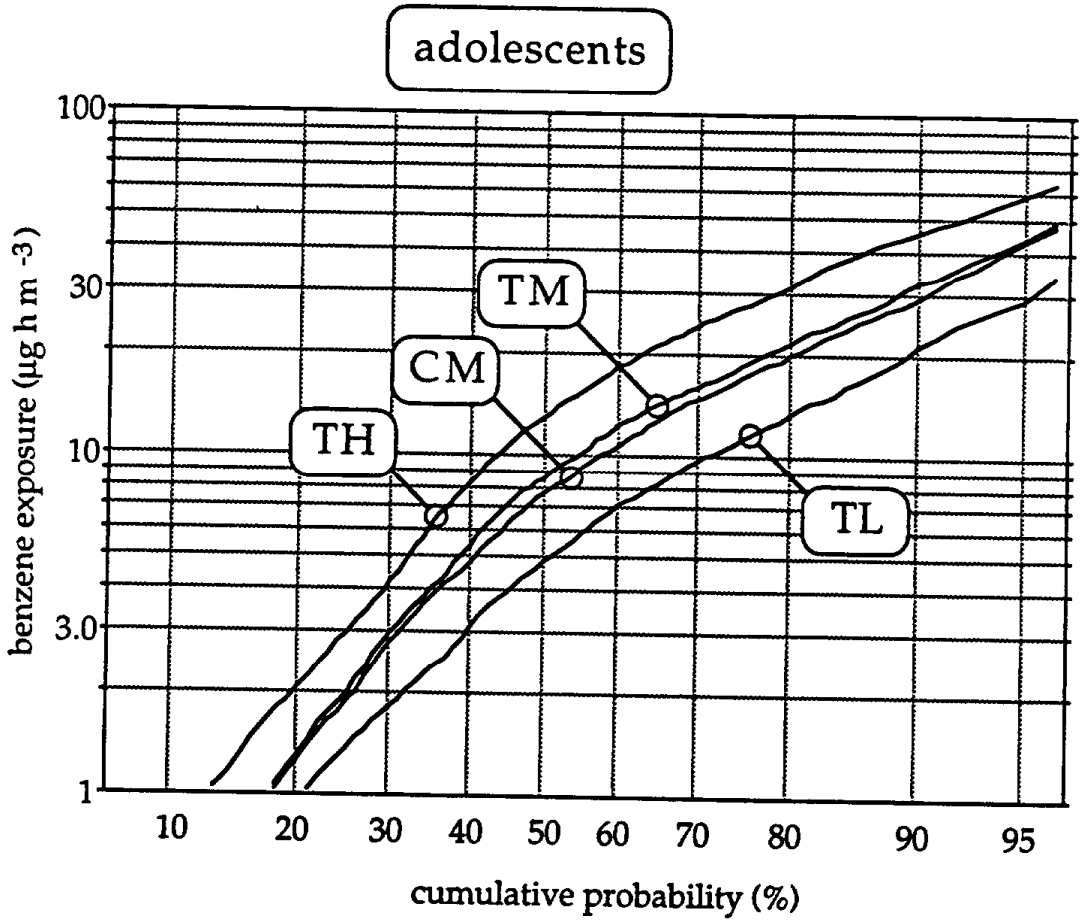

Figure 6.5b Lognormal-probability plot of the distributions of total exposure to benzene from ETS, separated by scenario, predicted for late 1990's for nonsmoking Californian adolescents. (Scenarios: TL - tracer low-range, TM - tracer mid-range; CM - completely mixed room model; TH - tracer high-range.)

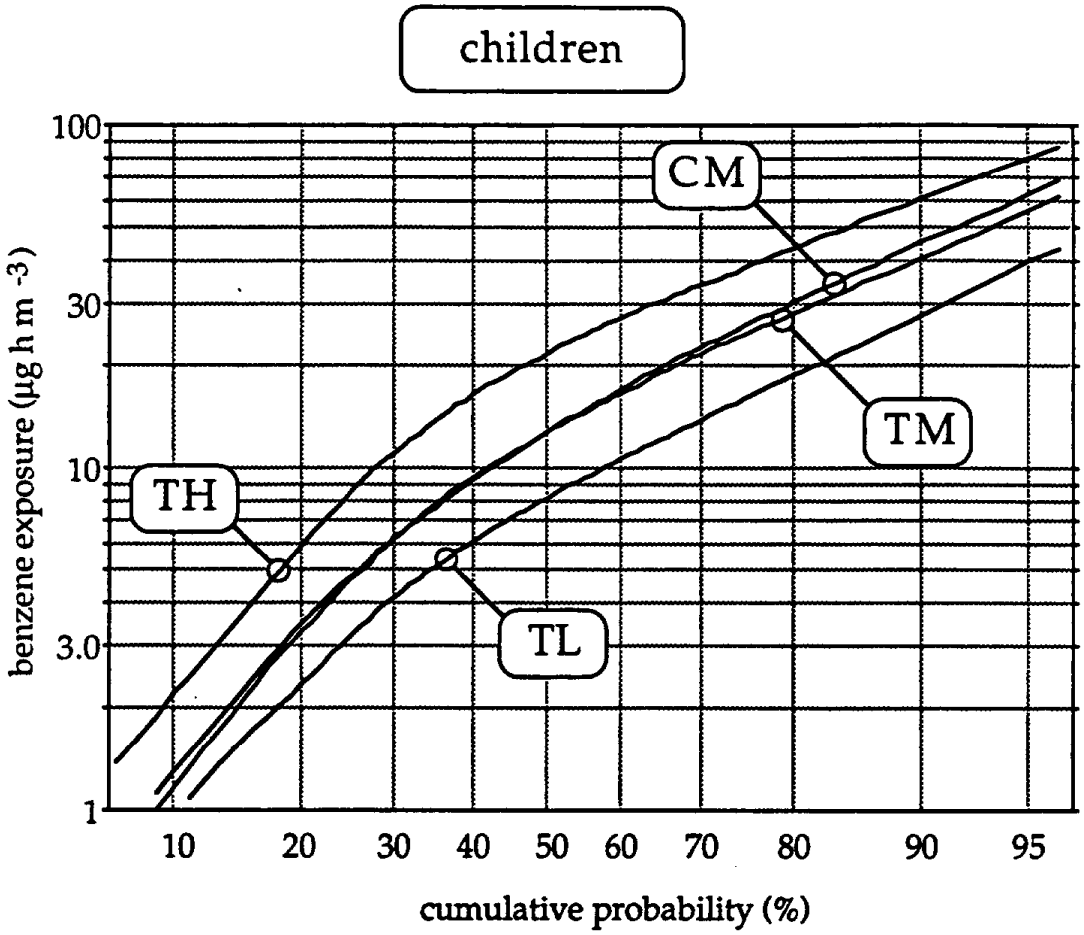

Figure 6.5c Lognormal-probability plot of the distributions of total exposure to benzene from ETS, separated by scenario, predicted for late 1990's for Californian children. (Scenarios: TL - tracer low-range, TM - tracer mid-range; CM - completely mixed room model; TH - tracer high-range.)

Differences among the four scenarios are relatively small compared with the variability in exposure across the population for any scenario. Largely, this reflects that the range of inputs for the tracer scenarios (TL, TM, and TH) for the residential microenvironment are small. Also, only one simulation approach with one set of parameters is used to model the transportation microenvironment. The true uncertainty in exposure for Phase III conditions may be larger than indicated by the range of scenario results. Nevertheless, it is reassuring that the CM scenario agrees well with the TM scenario (for adolescents and children) or the TL scenario (for adults) since, as in Phase II, these scenarios rely on independent methods for estimating microenvironmental ETS concentrations.

6.2.2.2 Contributions of Microenvironments

Figures 6.6-6.8 show the apportionment of the arithmetic mean (AM) exposure to ETS among different microenvironments for each of the scenarios. With the virtual elimination of exposures in occupational settings, as well as restaurants and bars, the apportionment of exposures among microenvironments varies much less among population groups than in Phase II. For all groups, the dominant site of average exposure is one's own home, contributing 58-69% of the total for adults, 58-66% for adolescents, and 72-83% for children.

Tables 6.5-6.7 and Figure 6.9 present greater detail on the contribution of each microenvironment to exposure. The tables present summary statistics for the distribution of exposures to benzene from ETS in each microenvironment. The results in this table only apply to those exposed in that particular setting, with the percentages of the nonsmoking population so exposed shown in Figure 6.3. Table 6.5-6.7 show that the ratio of the maximum to minimum scenario means in the residential (home or guest) microenvironments is approximately 2-3. For each scenario, variability in exposure across the population within a microenvironment is much larger. For example, Figure 6.9 shows that the ratio of C_{90} to C_{10} for residential exposures varies by a factor of 8-10. The variability of exposure among those exposed is even larger when total daily exposures in all microenvironments are considered (see Figure 6.5).

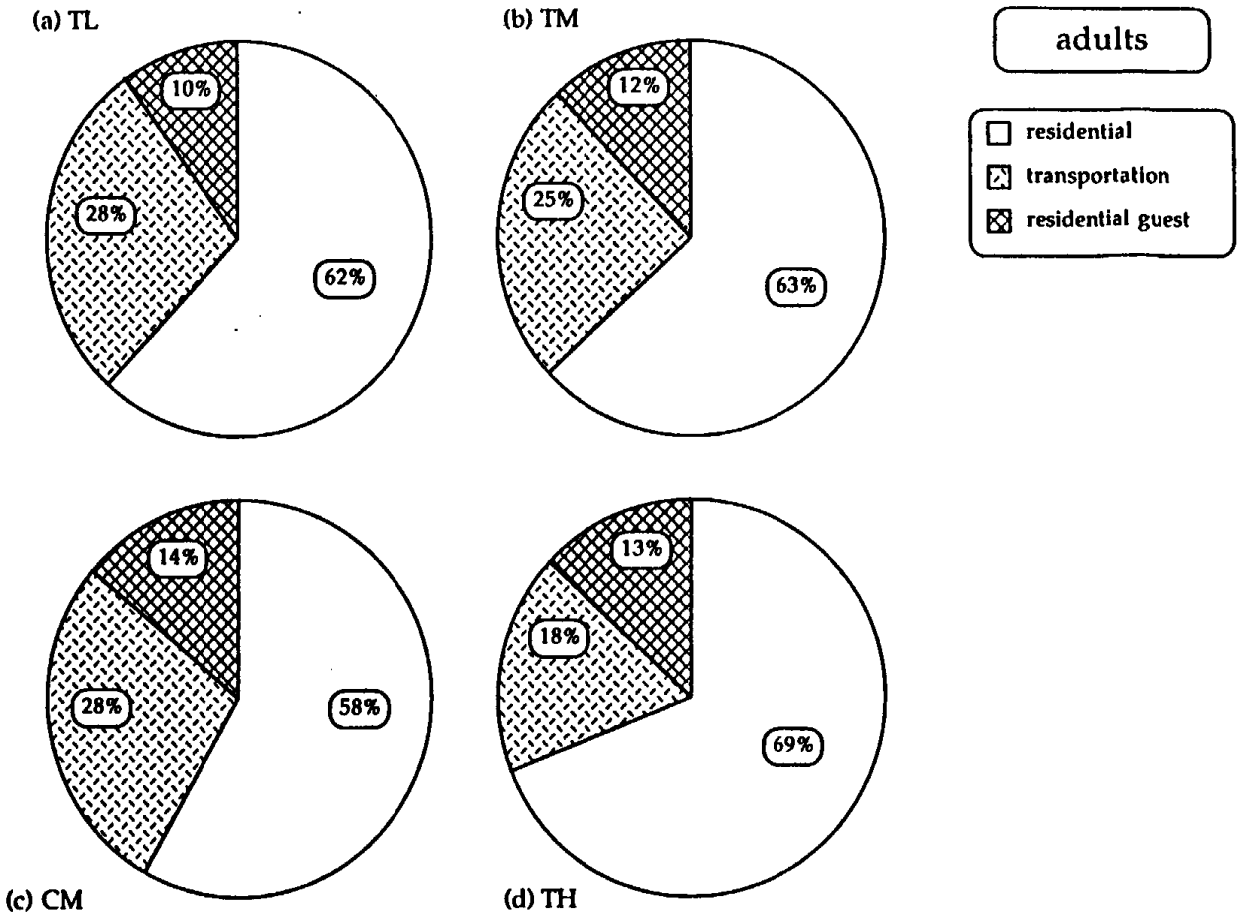

Figure 6.6 Proportion of arithmetic mean ETS exposure (late 1990's) among microenvironments for each of the four scenarios, for adult nonsmokers: (a) TL (tracer low-range), (b) TM (tracer mid-range), (c) CM (completely mixed room model), and (d) TH (tracer high-range)

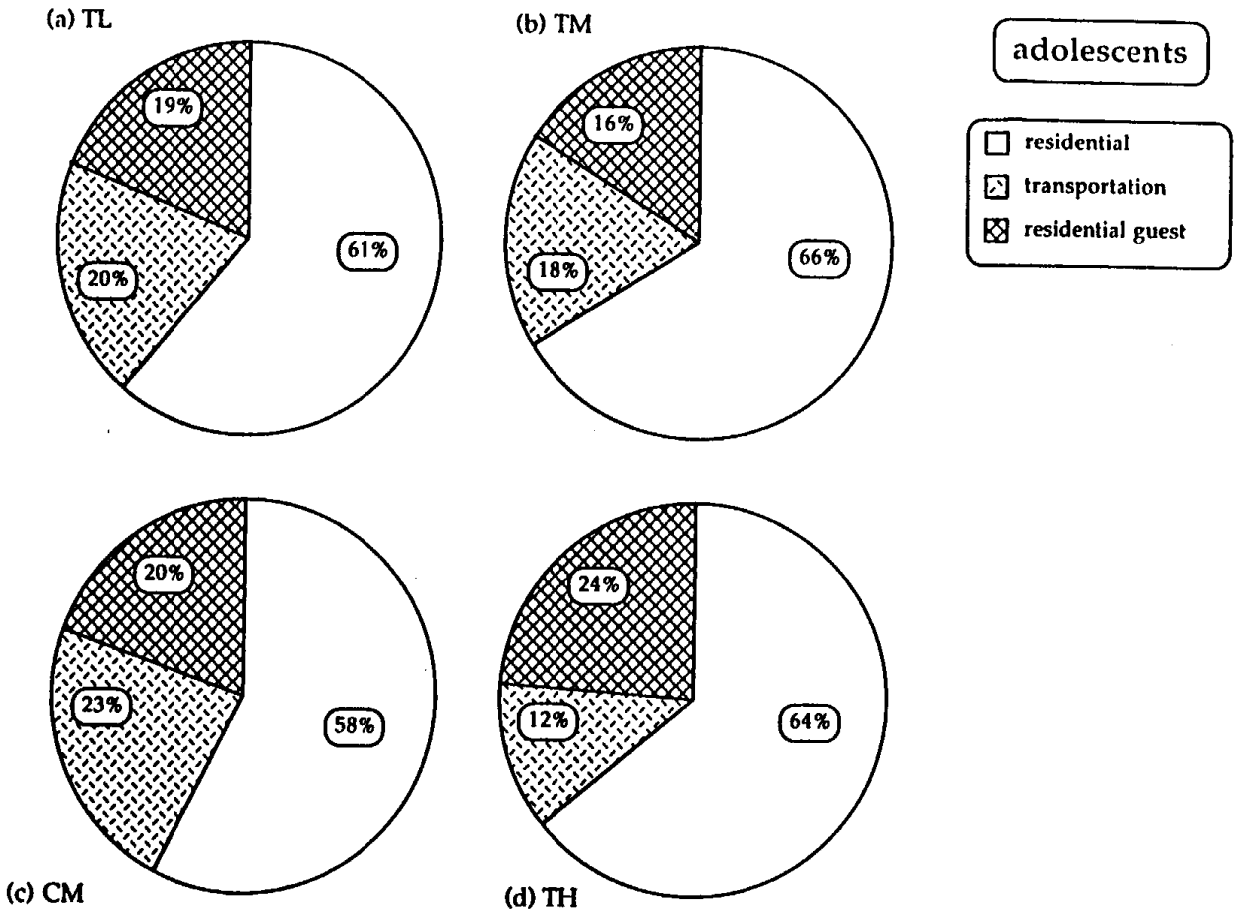

Figure 6.7 Proportion of arithmetic mean ETS exposure (late 1990's) among microenvironments for each of the four scenarios, for adolescent nonsmokers: (a) TL (tracer low-range), (b) TM (tracer mid-range), (c) CM (completely mixed room model), and (d) TH (tracer high-range)

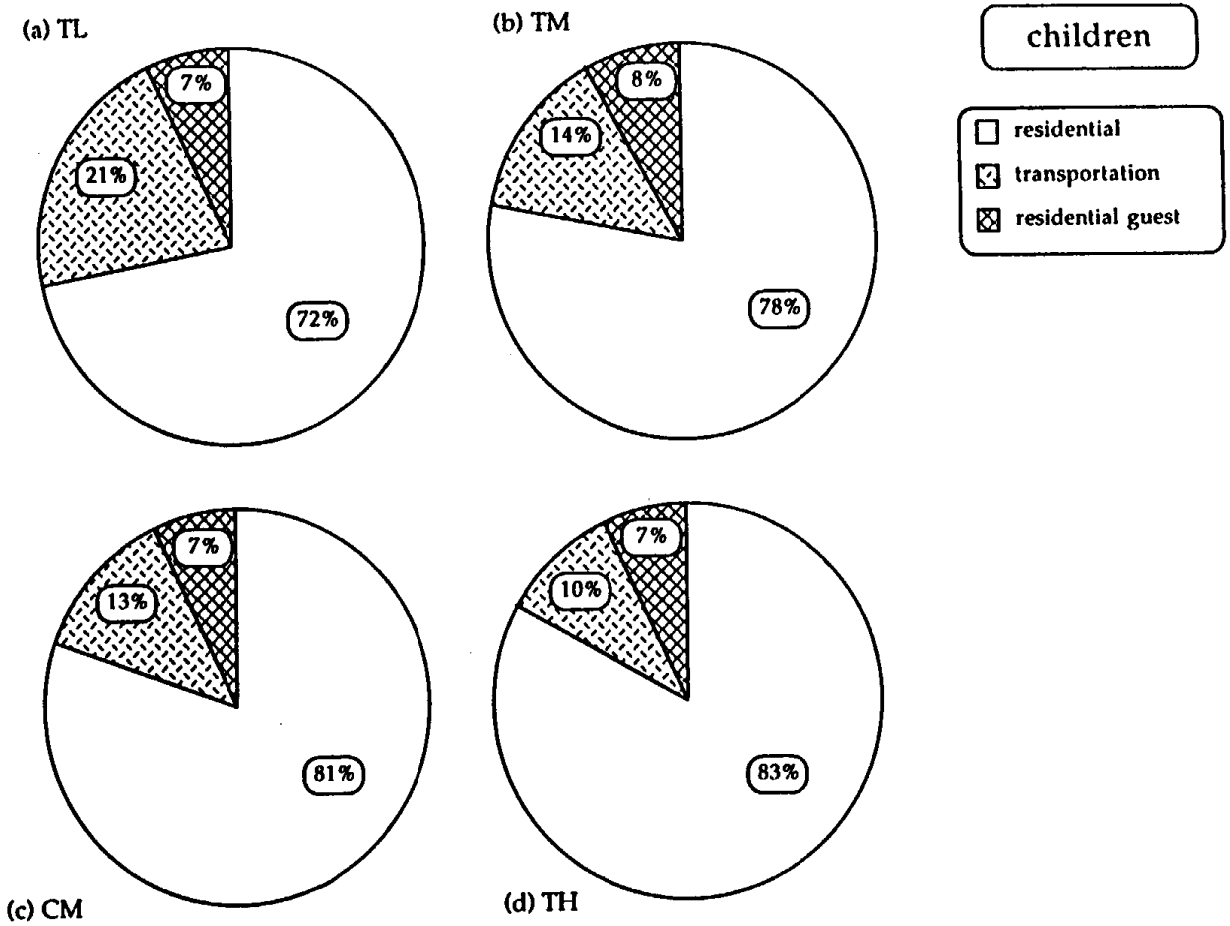

Figure 6.8 Proportion of arithmetic mean ETS exposure (late 1990's) among microenvironments for each of the four scenarios, for children: (a) TL (tracer low-range), (b) TM (tracer mid-range), (c) CM (completely mixed room model), and (d) TH (tracer high-range)

Table 6.5. Statistical parameters for daily exposure of the Californian adult nonsmoking population to benzene from environmental tobacco smoke in different microenvironments, predicted for late 1990's.[1,2]

microenvironment	*AM ($\mu g\ h\ m^{-3}$)*	*SD ($\mu g\ h\ m^{-3}$)*	*GM ($\mu g\ h\ m^{-3}$)*	*GSD*
residential				
tracer method				
low exposure	16	16	11	2.3
mid-range	20	19	15	2.2
high exposure	31	24	24	2.1
CMR method[3]				
mid-range	16	15	11	2.4
transportation				
CMR method[3]				
low exposure	9.2	26	2.7	5.0
mid-range	10	28	2.8	5.3
high exposure	9.9	24	3.1	5.1
residential guest				
tracer method				
low exposure	4.0	6.5	1.6	4.5
mid-range	5.3	11	1.8	4.7
high exposure	8.0	13	3.5	3.9

[1] AM–arithmetic mean, SD–arithmetic standard deviation, GM–geometric mean, GSD–geometric standard deviation.
[2] Results apply to that portion of the adult nonsmoking population in California that report some exposure in that microenvironment during a day (see Figure 6.3).
[3] CMR - completely mixed room model.

Table 6.6. Statistical parameters for daily exposure of the Californian adolescent nonsmoking population to benzene from environmental tobacco smoke in different microenvironments, predicted for late 1990's.[1,2]

microenvironment	*AM ($\mu g\ h\ m^{-3}$)*	*SD ($\mu g\ h\ m^{-3}$)*	*GM ($\mu g\ h\ m^{-3}$)*	*GSD*
residential				
tracer method				
low exposure	10	10	7.1	2.4
mid-range	17	18	12	2.5
high exposure	26	22	20	2.2
CMR method [3]				
mid-range	12	15	7.6	2.9
transportation				
CMR method [3]				
low exposure	4.8	7.2	2.1	4.2
mid-range	6.4	12	2.4	4.6
high exposure	6.8	12	2.5	4.6
residential guest				
tracer method				
low exposure	4.4	19	1.5	3.5
mid-range	5.6	13	1.9	4.6
high exposure	9.6	19	4.3	3.8

[1] AM–arithmetic mean, SD–arithmetic standard deviation, GM–geometric mean, GSD–geometric standard deviation.

[2] Results apply to that portion of the adolescent nonsmoking population in California that report some exposure in that microenvironment during a day (see Figure 6.3).

[3] CMR - completely mixed room model.

Table 6.7. Statistical parameters for daily exposure of the Californian children population to benzene from environmental tobacco smoke in different microenvironments, predicted for late 1990's.[1,2]

microenvironment	*AM ($\mu g\ h\ m^{-3}$)*	*SD ($\mu g\ h\ m^{-3}$)*	*GM ($\mu g\ h\ m^{-3}$)*	*GSD*
residential				
tracer method				
low exposure	13	16	9.1	2.3
mid-range	21	23	14	2.5
high exposure	31	27	23	2.2
CMR method [3]				
mid-range	22	27	13	2.8
transportation				
CMR method [3]				
low exposure	6.7	16	2.4	4.5
mid-range	7.2	19	2.5	4.9
high exposure	7.6	17	2.7	4.7
residential guest				
tracer method				
low exposure	3.6	6.1	1.3	5.0
mid-range	6.0	11	2.0	5.3
high exposure	9.3	14	3.7	4.7

[1] AM–arithmetic mean, SD–arithmetic standard deviation, GM–geometric mean, GSD–geometric standard deviation.
[2] Results apply to that portion of children in California that encounter some exposure in that microenvironment during a day (see Figure 6.3).
[3] CMR - completely mixed room model.

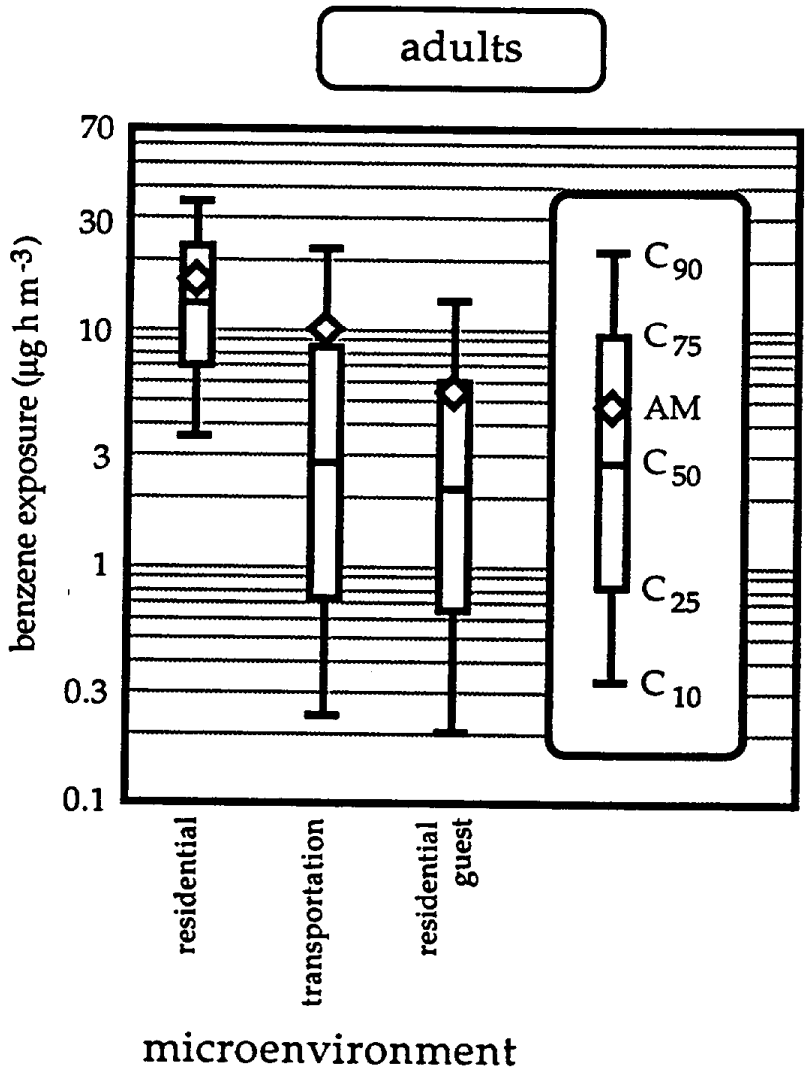

Figure 6.9a Whisker diagram for benzene exposure variability from ETS among microenvironments in scenario CM (completely mixed room model), predicted for late 1990's for nonsmoking adults. (AM is the arithmetic mean and C_i represents the i^{th} percentile of the distribution.)

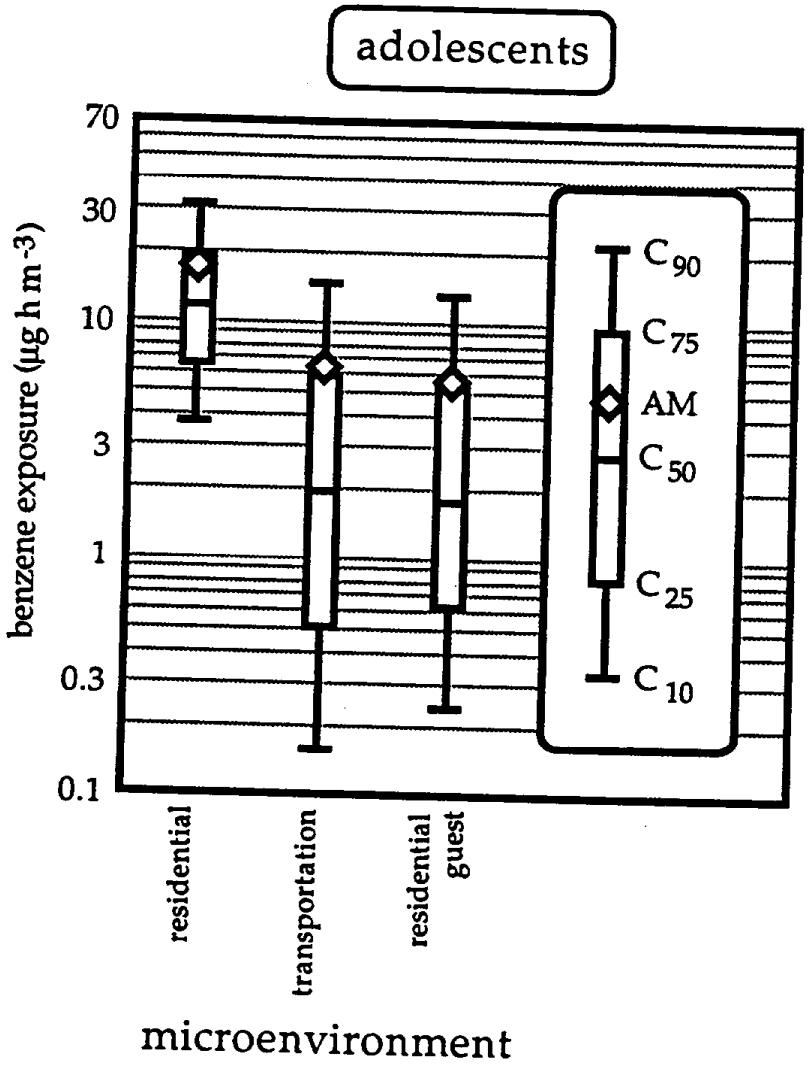

Figure 6.9b Whisker diagram for benzene exposure variability from ETS among microenvironments in scenario CM (completely mixed room model), predicted for late 1990's for nonsmoking adolescents. (AM is the arithmetic mean and C_i represents the i^{th} percentile of the distribution.)

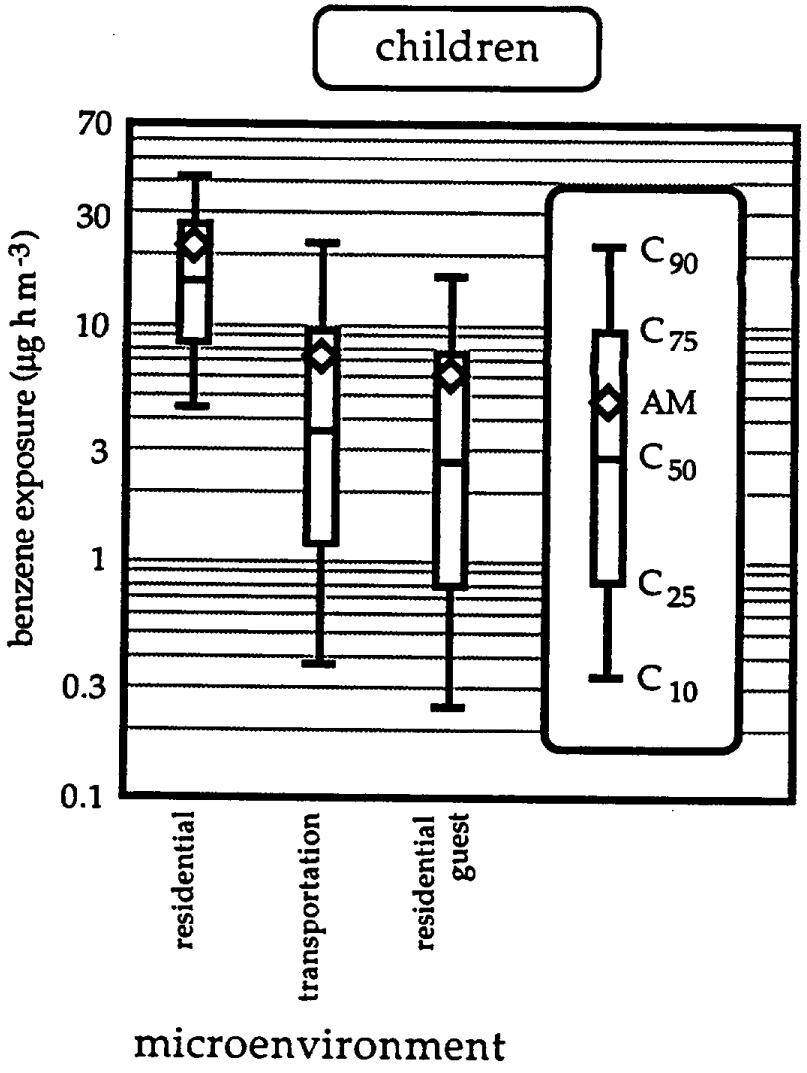

Figure 6.9c Whisker diagram for benzene exposure variability from ETS among microenvironments in scenario CM (completely mixed room model), predicted for late 1990's for children. (AM is the arithmetic mean and C_i represents the i^{th} percentile of the distribution.)

6.2.3 Predicted Mean Exposure for All Nonsmokers to TACs from ETS

Using the arithmetic mean values for each scenario, we predicted the contribution of ETS to the average exposure of nonsmoking Californians for the seventeen air toxicants considered in this study. Table 6.8 presents these results, along with a summary of results from Phase II. As before (see Table 5.9), to estimate mean exposure for the entire nonsmoking population, we multiplied the estimates of mean exposure for those exposed by the fraction of the nonsmoking population that was exposed in each evaluation.

On a per-capita basis (i.e., averaged over all nonsmokers in a given age group), mean exposures of nonsmoking adolescents and children are predicted to be higher than mean exposures for nonsmoking adults. For benzene, for example, Table 6.8 shows that the average contribution of ETS to children's daily exposure is 2.5-6.5 $\mu g\ h\ m^{-3}$, higher than the 2.0-4.0 $\mu g\ h\ m^{-3}$ range predicted for adults. Considered one scenario at a time, for children, the predicted population mean exposure to air toxics from ETS ranges from 130% (scenario TL) to 190% (scenario CM) of that predicted for adult nonsmokers. Much of the increased exposure for children results from the higher incidence of exposure (21-23% vs. 16-19%). For nonsmoking adolescents, the range of population mean exposures is predicted to be 150% (scenario TL) to 200% (scenario TH) of that for nonsmoking adults. Again, the difference is primarily a consequence of the higher predicted incidence of exposure among adolescents (33-35% vs. 16-19%).

6.2.4 Changes in Exposure from late 1980's to late 1990's

Figure 6.10 presents a summary of the Phase II and Phase III results, emphasizing the differences in exposure among population age groups and the changes that have occurred between the late 1980's and late 1990's in California. Each bar in the figure represents the normalized exposure to ETS of a population group according to one of the scenarios in one of the two time periods. The height of the bar is determined by multiplying the appropriate mean benzene exposure (Tables 5.3-5.5 or Tables 6.2-6.4) by the fraction of the population group exposed, and then dividing by the corresponding results for adult nonsmokers in the late 1980's. This figure shows that in the late 1980's, the predicted per-capita mean exposure of adolescent nonsmokers was in the range of 80-120% of the corresponding mean for adult nonsmokers. For children, the normalized average exposure was smaller: 50-80% of the mean adult exposure.

Table 6.8. Comparison of Phase II (late 1980's) and Phase III (late 1990's) estimates of average daily exposure of all nonsmoking Californians to toxic air contaminants from ETS.

	ETS-only, daily exposure ($\mu g\ h\ m^{-3}$)					
	Phase II [1]			*Phase III* [2]		
compound	*adults*	*adolescents*	*children*	*adults*	*adolescents*	*children*
acetaldehyde	25-83	30-67	21-43	10-21	16-42	13-35
acetonitrile	20-68	23-52	16-32	8-16	12-32	10-26
acrylonitrile	1.2-4.0	1.4-3.1	1.0-1.9	0.5-1.0	0.7-1.9	0.6-1.6
benzene	4.8-16	5.6-13	4.0-7.9	2.0-4.0	3.0-7.7	2.5-6.5
1,3-butadiene	1.8-6.2	2.1-4.7	1.5-3.0	0.7-1.5	1.1-2.9	0.9-2.4
2-butanone (MEK)	3.5-11	4.0-9.0	2.8-5.6	1.4-2.9	2.1-5.6	1.8-4.6
o-cresol	0.4-1.4	0.5-1.1	0.3-0.7	0.2-0.3	0.3-0.7	0.2-0.6
m,p-cresol	1.0-3.3	1.1-2.6	0.8-1.6	0.4-0.8	0.6-1.6	0.5-1.3
ethyl acrylate	< 0.12	< 0.10	< 0.06	< 0.03	< 0.06	< 0.05
ethylbenzene	1.6-5.1	1.8-4.0	1.3-2.5	0.6-1.3	1.0-2.5	0.8-2.1
formaldehyde	16-52	18-41	13-25	6-13	10-25	8-21
n-nitrosodimethylamine	0.007-0.023	0.008-0.018	0.006-0.011	0.003-0.006	0.004-0.011	0.004-0.009
phenol	3.3-11	3.9-8.8	2.7-5.6	1.4-2.9	2.1-5.3	1.7-4.4
styrene	1.8-5.7	2.0-4.5	1.4-2.9	0.7-1.4	1.1-2.8	0.9-2.3
toluene	7.8-26	8.8-20	6.3-13	3.1-6.5	5.0-13	4.0-10
o-xylene	0.8-2.7	1.1-2.1	0.7-1.3	0.3-0.7	0.5-1.3	0.4-1.1
m,p-xylene	3.5-12	4.2-9.5	2.9-5.9	1.4-2.9	2.2-5.6	1.8-4.9

[1] Range of arithmetic means for scenarios TL, TM, CM, and TH; obtained by multiplying appropriate values in Tables 5.3-5.5 by percentage of nonsmokers exposed to ETS on a daily basis in simulated microenvironments (52% for adults, 63% for adolescents, and 33% for children).

[2] Range of arithmetic means for scenarios TL, TM, CM, and TH; obtained by multiplying appropriate values in Tables 6.2-6.4 by percentage of nonsmokers estimated to be exposed to ETS on a daily basis for late 1990's in simulated microenvironments (Table 6.1).

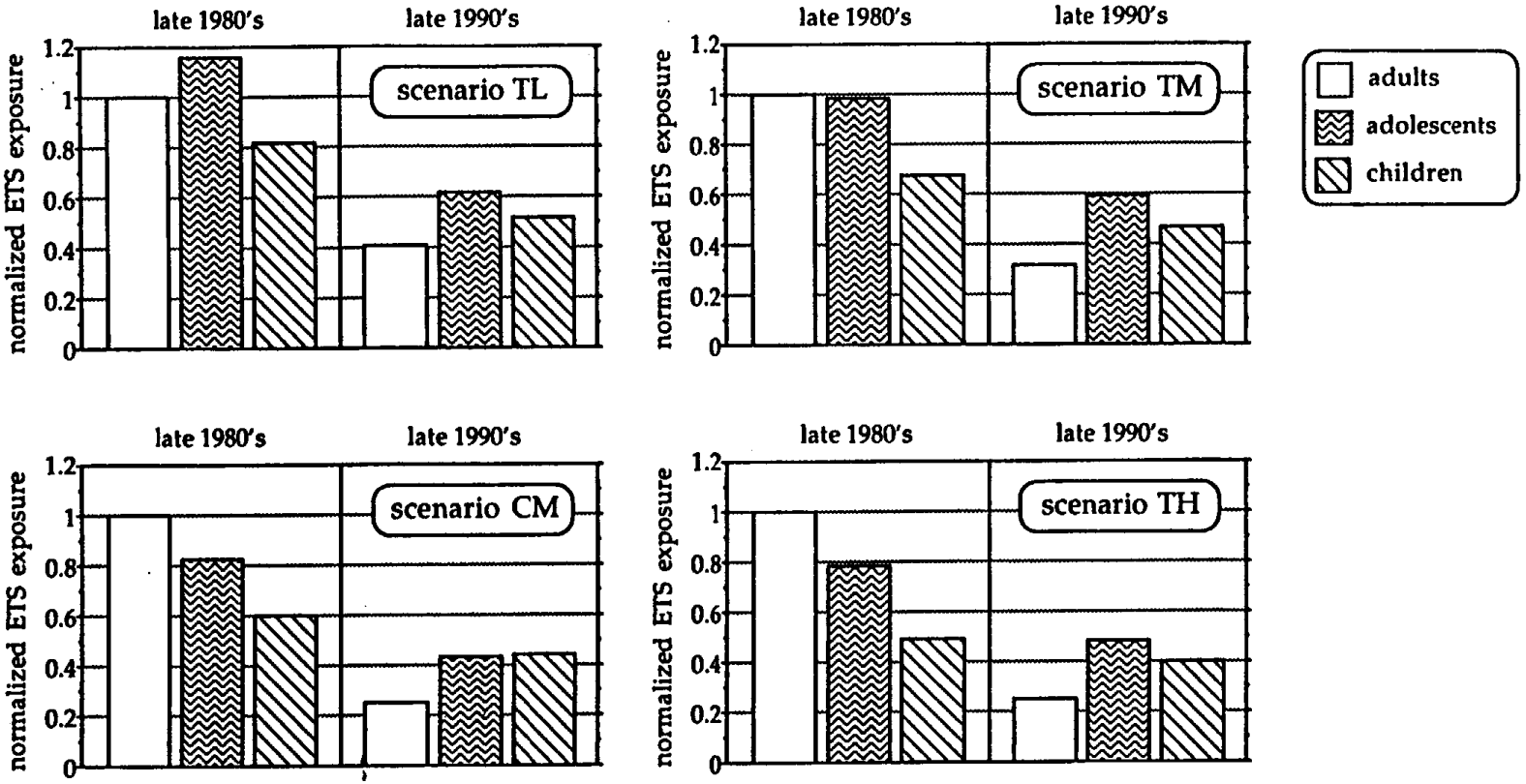

Figure 6.10 Normalized average exposure of all nonsmokers to ETS, segregated according to scenario, research phase (Phase II = late 1980's and Phase III = late 1990's), and population age group. For each scenario, the exposure is normalized so that the value for adults in the late 1980's is unity. (Scenarios: TL - tracer low-range, TM - tracer mid-range; CM - completely mixed room model; TH - tracer high-range.)

In the late 1990's, predicted mean exposures have declined. The decline is greatest for adults: their exposures are reduced to 25-40% of the estimated values for the late 1980's. The mean exposure for adolescents and children are also predicted to be smaller in the late 1990's than they were in the late 1980's. However, they are predicted to be higher than the mean for adults in the late 1990's, in the range 45-60% of the late 1980's adult mean for adolescents and 40-50% for children.

Overall, the predicted reduction occurs because of two factors: the elimination of smoking in most public buildings because of AB13 and the reduced prevalence of adult smoking. The former factor has a large effect on adult exposures, but less for adolescents and especially children. The latter factor benefits all population groups roughly equally.

6.2.5 Assessment Limitations

Since the same modeling methods were applied in Phase III as in Phase II, most of the limitations that were discussed in Section 5.3.2 also hold for Phase III.

Relative to the Phase II assessment, there are some aspects of Phase III that reduce uncertainty and others that increase uncertainty. In Phase II, significant contributions to ETS exposure occurred in the "retail/other" group of microenvironments. Only sparse data are available to predict exposures in those settings, and that fact contributes substantially to uncertainty in the Phase II results. By contrast, because AB13 effectively bans smoking in those settings, not only are the exposures in Phase III expected to be very small, but the uncertainty in exposure (e.g., caused by violations of the regulation) is also expected to be small. On the other hand, the activity pattern data applied in both Phase II and Phase III assessments were collected in the late 1980s. The sampling period coincides well with the target time period of the Phase II assessment, but is about a decade earlier than the target time period in Phase III. Any temporal changes in the activities of Californians contributes to uncertainty in the Phase III assessment. Similarly, much of the data used to predict microenvironmental concentrations of ETS constituents was gathered in the 1980s (see Table 5.2) and uncertainty is introduced in extrapolating to the late 1990s.

In carrying out the both the Phase II and Phase III assessments, we have sought to avoid making assumptions that significantly bias the exposure estimates. At the same time, we have aimed to base the evaluations as much as possible on parameters whose values are derived from appropriate data rather than assumed. The net effect is that some residual bias may remain in the outcome, but such bias is not expected to be large.

Some effects are expected to lead to an underestimate of exposure. For example, in the transportation microenvironment we assumed that only one smoker was active during a given exposure episode. Since, by definition, an exposure episode requires a minimum of one smoker and more than one smoker is possible, this assumption will bias the results towards

underestimating exposure. Similarly, the contribution of outdoor smoking to ETS exposure was ignored in our assessment. Since some exposure occurs as a result of outdoor smoking, either because of a nonsmokers proximity to a smoker or because the smoker is close to a building ventilation intake, the population mean exposure to ETS from smoking outdoors is greater than zero. Also, we have not included any residual exposures that occur in workplaces following implementation of AB13. Some exposure could still occur in workplaces where AB13 is violated, or in the few cases where smoking is still permitted by AB13. Californians may also be exposed to ETS at higher than predicted rates when they travel outside the state because of more permissive smoking regulations and because of a higher prevalence of smoking elsewhere. That these biases exist is not questioned. The important issue is whether they are significant contributors to estimated mean exposures and to the estimated variance in population exposure. In our judgment, these effects are not likely to be large.

In other respects the assessment method may be biased to overestimate ETS exposure. A primary factor here may be behaviors practiced either by smokers or nonsmokers that are deliberately intended to reduce ETS exposure. In residences, for example, people may step outside to smoke, or may smoke in a separate room with doors closed and a window open or an exhaust fan operating. When smoking in a vehicle, it is common for a smoker to open their window and position the cigarette so that the sidestream smoke doesn't all enter the passenger compartment. Some of these practices may be captured in our modeling assessment. For example, ETS tracer measurements in smokers homes would reflect indoor concentrations as they occur, implicitly incorporating information about smokers' behavior that influenced the concentrations. However, not all such practices are captured in the model (e.g., window operation in a vehicle with a smoker). Furthermore, a cultural shift in the degree to which smoking is accepted seems to be occurring. It appears that the idea that a nonsmoker has the right to not be exposed to tobacco smoke is becoming more accepted. If so, it seems quite possible and perhaps even probable that personal-choice behaviors intended to reduce nonsmoker exposure to ETS are more widely practiced in the late 1990s than they were in the 1980s. The modeling approach used in Phase III does not account for such effects. As with the issues that would tend to bias the results towards underestimating exposure, not enough is known to quantify the effect of these factors that would tend to bias the results towards overestimating exposure.

By the design of our research methods, the uncertainty in mean exposure is indicated by differences among scenarios. Among those exposed in the Phase III assessment, the ratios of TH to TL scenario means are 1.7 for adults, 2.4 for adolescents, and 2.3 for children (see Tables 6.2-6.4). The variability in exposure, indicated by the ratio of the 90th to the 10th percentile exposure concentration among those exposed, ranges from roughly a factor of 30 for children to a factor of

70 for adults and adolescents (see Figure 6.4). Relative to these indicators, we believe that the effect of biases on the results of the Phase III modeling is not large.

7. Summary and Conclusions

As presented in the introduction, we pursued five objectives in this project. This section summarizes how the objectives were pursued and what findings resulted.

Throughout, the research focused on nonsmoking Californians. Three separate population age groups were considered: adults (aged ≥18 y), adolescents (12-17 y), and children (0-11 y). (In Phase I, the assessment was conducted for all nonsmokers ≥7 y old without segregation into separate age groups.)

Exposures were quantified on the basis of either a daily total (in units of $\mu g\ h\ m^{-3}$) or an incremental exposure concentration ($\mu g\ m^{-3}$). The former represents the accumulated exposure over a 24-h period (the time integral of the concentration to which one is exposed). The latter represents the time-averaged increase in the concentration to which one is exposed to a contaminant from ETS.

7.1 Frequency Distribution of Exposure to TACs from ETS, late 1980's

Two methods were applied to estimate the frequency distribution of exposure across the California population. In Phase I of the research, the results of personal monitoring studies were analyzed to infer the contribution of ETS to exposure. In these studies, the time-weighted average exposure concentrations of many volatile organic compounds were measured for statistically selected subjects from different regions of California. The responses of the subjects to administered questionnaires permitted us to distinguish those exposed from ETS during the measurement period from those unexposed (and also to eliminate active smokers from our study group). We developed and applied a probabilistic simulation method to infer the exposure from ETS based on differences in the distributions of exposures of exposed and unexposed subjects. The method was separately applied for four species: benzene, styrene, o-xylene, and m,p-xylene. Based on the average results for these four species, and utilizing emission factor data, exposure predictions were also made for 13 other air toxics. The principal results were presented in Table 4.2, which reports the estimated population mean exposure from ETS for those exposed. Variances in exposure were also estimated.

In Phase II, a second method was developed to pursue the same objective. In this case, exposures were estimated by combining data on the activity patterns of California residents with estimates of microenvironmental concentrations. Concentrations were determined from measurements of tracers of tobacco smoke — nicotine and particulate matter — and from material balance models. Using separate activity data for adults, adolescents, and children, it was possible to estimate the exposures of these population groups separately. Results from this effort are

summarized in Table 5.9 which shows the estimated average daily exposure of all California nonsmokers to 17 toxic air contaminants from ETS, both as predicted in Phase I and in Phase II. Variability in exposures within population groups is best revealed in Figures 5.1 and 5.2.

7.2 Proportion of Exposure from ETS

The average proportion of nonsmokers exposure caused by ETS could be estimated for four compounds — benzene, styrene, o-xylene, and m,p-xylene. These results were computed as the ratio of two numbers: the numerator was an appropriate result from this study; the denominator was based on the measured average exposure of nonsmokers from the personal monitoring studies used in Phase I (as summarized in Table 4.1). Table 4.3 shows the proportion of exposure attributable to ETS as predicted in Phase I, for exposed nonsmokers and also for the entire nonsmoking population. Figure 5.7 displays results in graphical form, considering only the population exposed, but including Phase II results along with Phase I. Among those exposed, Figure 5.7 shows that the average contribution of ETS to total exposure is in the range 3-10% for benzene, 6-19% for styrene, 0.5-8% for o-xylene, and 1-5% for m,p-xylene. The Phase I results show excellent agreement with the Phase II findings for benzene and styrene. The agreement is fair for m,p-xylene. For o-xylene, the Phase I predictions (8% contribution to exposure) are substantially higher than the Phase II predictions (0.5-2% contribution to exposure).

For compounds other than these four, the proportion of exposure from ETS could not be estimated, because the total personal exposure is unknown.

7.3 Relative Amounts of Exposure in Different Microenvironments

The Phase II approach permits us to estimate the contribution of different microenvironments to total exposure, and this goal was pursued. Results are best summarized by the pie charts in Figure 5.3-5.5, which show the fraction of mean ETS exposure that occurs in different microenvironments for different population age groups, estimated for the late 1980's. From Figure 5.3, we see that substantial exposure of adults occurred in many different microenvironments. The largest contributions came from residential (19-41%) and occupational (20-39%) settings. However, significant contributions also occurred in other microenvironments included in the study: restaurants, bars and nightclubs, transportation, retail/other indoor, and residential guest. For adolescents, exposure in ones own residence assumes a larger proportion of the total (48-58%) and occupational exposure is insignificant (Figure 5.4). For children, personal residences overwhelm other microenvironments as a site of ETS exposure, contributing 70-73% of the total (Figure 5.5).

7.4 Impact of Changes in Policy and Behavior on Exposure for late 1990's

The implementation of state legislation that severely limits smoking in public buildings (AB13) accompanied by aggressive intervention efforts to reduce the prevalence of smoking are expected to have a substantial effect on the exposure of nonsmokers to environmental tobacco smoke. In Phase III of the project, we applied the modeling approach developed in Phase II with input intended to represent current conditions to predict the exposure of nonsmokers to air toxicants in ETS for the late 1990's. The overall results of this effort were presented in detail in §6 of the report. The most succinct summary is presented in Figure 6.10 which shows the population mean ETS exposure for different population age groups, normalized to the population mean for adults in the 1980's. ETS exposures are predicted to be substantially lower for all groups in the late 1990's than they were a decade earlier. According to the predictions, this has occurred primarily because smaller proportions of the population are exposed. Because policy changes reflected in AB13 focus on workplaces, where only a small fraction of children's and adolescent's ETS exposure occurred, the predicted exposure reductions for adults are much larger than for juveniles. In fact, modeling results indicate that although the per-capita exposure of children to ETS was considerably smaller than adult exposure in the late 1980's, it is substantially higher than adult exposure now.

7.5 Critique Quality of Results

We have devoted much attention, throughout this research, to assessing the uncertainty in our estimates. In Phase I, a method was developed and applied to explicitly evaluate confidence intervals about the central estimates of exposure. The method was based on computational experiments and focused on what we judged to be a primary source of uncertainty — the relatively small size of the population studied. We found that the uncertainty in the estimated mean exposure was relatively large. The 90% confidence intervals are a factor of $\times/\div$ 4 from the central estimate for styrene and o-xylene, $\times/\div$ 6 for benzene and $\times/\div$8 for m,p-xylene. To a great degree, these large uncertainties reflect the fact that we were trying to quantify small fractional differences in mean exposure between those exposed to ETS and those unexposed when both population groups exhibited high variability.

In Phases II and III of the research, uncertainties do not appear to be as large. We attempted to approximately bracket the range of possible outcomes (without exaggerating the uncertainty) by constructing four distinct scenarios. In two of the four scenarios, microenvironmental concentration estimates for most simulated settings were selected deliberately to be either at the low or high end of reported applicable values. The range of results between the low-range and high-range scenarios is a factor of approximately 2-3, and this is a fair indicator of the uncertainty in the predicted mean exposures in these phases of the research.

In addition to the efforts to quantify uncertainty in each phase of the research, we have constructed the approaches so that they complement one another. For example, the Phase I evaluation of exposure from ETS to benzene, styrene, o-xylene, and m,p-xylene is *completely independent* of the Phase II evaluation of exposure to these species in the sense that neither the input data nor the methods were common between the two efforts. The general agreement in mean exposure estimated by the two methods adds confidence to the results, especially for benzene and styrene. Furthermore, the CM scenario in Phases II and III used an independent method for estimating microenvironmental concentrations relative to the T- scenarios for the two most important microenvironments — residences and occupational settings. The agreement between the CM and TM (or TL) scenarios is generally very good, as revealed by Tables 5.3-5.5, Tables 6.2-6.4, and Figures 5.2 and 6.5. This agreement provides further evidence that the accuracy of the estimates is at least as good as indicated by the uncertainty estimates.

7.6 Recommendations for Future Research

Research along any of several fronts would further improve our understanding of the exposure of Californians to toxic air contaminants from environmental tobacco smoke. Ideas that emerged from our study are discussed briefly here.

In the absence of any other information, we were obliged to assume that the activity patterns of California residents in the late 1980's still applied in the late 1990's. Demographic, socioeconomic, and other changes might have an influence on activities that would in turn affect the frequency and duration of ETS exposure activities. Future assessments of human exposure to air pollutants would benefit from the design and execution of an updated set of activity pattern surveys.

If another survey of activity patterns were undertaken with the intent to apply the results in assessing ETS exposure, then additional information related to tobacco use should be gathered. The existing surveys, while constituting rich sources of data about the frequency and duration of exposure, were notably weak in providing information about the *intensity* of exposure. Data on the number of smokers who live in a household, the number of cigarettes smoked in one's residence, and the number of smokers and/or cigarettes smoked in one's presence in different settings would have been invaluable in conducting our assessment.

Another worthwhile research topic would be to measure ETS emission factors for other toxic compounds. Cigarette smoke is a known source of toxic metals, such as lead, cadmium, and mercury (Rickert and Kaiserman, 1994), but ETS emission factors for these elements are lacking. Tobacco smoke also contains polycyclic aromatic hydrocarbons (Gmeiner et al., 1997). Polycyclic organic matter is listed collectively as a toxic air contaminant. The specific compounds considered in this study are all of the formally designated toxic air contaminants for which reliable data exist

on ETS emission factors. But because tobacco smoke is a source of other toxic compounds, the exposures quantified in this study are expected to cause only a portion of the total health hazard from ETS.

The analyses presented here are based on assumption that the concentrations of ETS constituents are present in approximately the same proportion, independent of environmental conditions. This assumption has been criticized in the context of using nicotine as a marker compound (Nelson et al., 1992). Recent research provides some limited support for the assumption (Van Loy et al., 1998). However, the assumption is central to much of the analyses presented here, and so additional explicit research to address the issue seems warranted. A useful set of experiments could be conducted, for example, in which smoking was habitually conducted in a test room, furnished as an ordinary residential room. The time-averaged concentrations would be measured for a suite of compounds in ETS, including volatile organic compounds, semivolatile compounds, ETS tracers, and particulate phase materials. The data would reveal to what extent the species concentrations are present in constant proportion in realistic, albeit controlled circumstances.

Another pertinent topic that could fruitfully be studied is the degree of compliance with the provisions of AB13. In the present assessment, we assumed that exposures associated with violations of AB13 would be negligible. The validity of that assumption might be effectively tested through a population survey, either as part of an updated activity pattern survey, or alternatively in connection with an ongoing survey such as California's Behavioral Risk Factor Survey.

Our study has shown that the distribution of ETS exposures differ among Californians according to age. Because of differences among population subgroups in factors such as smoking behavior, we also expect that ETS exposures might vary among Californians according to socioeconomic status, race, gender, and/or ethnicity. The methods applied in this study could be adapted to explore these issues. The results might be useful in the design of mitigation strategies to reduce exposure.

Lastly, additional monitoring of environmental tobacco smoke in different microenvironments would also help strengthen the quality of future assessments of ETS exposure. A well-designed study to measure the concentrations of environmental tobacco smoke tracers in a statistical sample of California residences and in vehicles in which smoking occurs could significantly reduce the uncertainty in future exposure assessments.

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9. Publication Resulting from this Research

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10. Glossary of Terms, Abbreviations, and Symbols

adolescents	age range 12-17 y
adults	age range ≥18 y
AM	arithmetic mean
APCR	Activity Patterns of California Residents
ARB	(California) Air Resources Board
C	species concentration ($\mu g\ m^{-3}$)
C'(t)	time-dependent variation of concentration relative to the mean ($\mu g\ m^{-3}$)
C_{avg}	time-averaged species concentration ($\mu g\ m^{-3}$)
$C_{benzene}$	concentration of benzene from ETS in a given microenvironment ($\mu g\ m^{-3}$)
$C_{nicotine}$	concentration of nicotine from ETS in a given microenvironment ($\mu g\ m^{-3}$)
C_{PM}	concentration of particulate matter from ETS in a microenvironment ($\mu g\ m^{-3}$)
C_{10}	concentration or exposure corresponding to 10th percentile in a distribution
C_{25}	concentration or exposure corresponding to 25th percentile in a distribution
C_{50}	concentration or exposure corresponding to 50th percentile in a distribution
C_{75}	concentration or exposure corresponding to 75th percentile in a distribution
C_{90}	concentration or exposure corresponding to 90th percentile in a distribution
CAP	Children's Activity Patterns
CED	Californian Exposures Database
CM	exposure scenario based on CMR model, using mid-range parameters
CMR	completely mixed room model
CO	carbon monoxide
COV	coefficient of variation (= SD/AM)
children	age range 0-11 y
D_{KS}	Kolmogorov-Smirnov statistic
e	emission factor for the air toxicant in ETS ($\mu g\ cig^{-1}$)
e_i	emission factor for compound i ($\mu g\ cig^{-1}$)
e_r	emission factor for reference compound r ($\mu g\ cig^{-1}$)
E	emission rate of contaminant into indoor air ($\mu g\ h^{-1}$)
ETS	environmental tobacco smoke
F_i	ith value in a probability distribution function
f	fraction of occupants that smoke (=n/Γ)
f(y)	true probability distribution function of exposure levels to a contaminant in ETS for nonsmokers who are exposed to ETS

$f_1(y^*)$	constructed estimate of f(y) (see Appendix C)
$\hat{f}(y)$	estimate of f(y) derived from CED
g(x)	true probability distribution function of exposure levels to a contaminant in ETS of nonsmokers who are unexposed to ETS
$\hat{g}(x)$	estimate of g(x) derived from CED
GM	geometric mean
GM_{ETS}	geometric mean exposure concentration to a toxic air contaminant from ETS only
GSD	geometric standard deviation
GSD_{ETS}	geometric standard deviation for distribution of exposure concentrations to a toxic air contaminant from ETS only
h(z)	probability distribution function for exposure levels to a contaminant in ETS for a hypothetical population of nonsmokers who are exposed only to ETS
$h_k(z)$	kth hypothesized estimate of h(z), where k is an integer
$\hat{h}(z)$	the best estimate of h(z)
MS	mainstream smoke; that which is inhaled by an active smoker
microenvironment	location or group of locations where exposure to an air pollutant occurs; typically the interior of a room, a building, or a vehicle
Monte-Carlo	simulation procedure that uses repeated random draws from one or more probability distribution functions to generate statistically accurate assessment
n	number of smokers in microenvironment
N_o	rate at which cigarettes are smoked in occupational setting (cig h^{-1} per smoker)
N_r	rate at which cigarettes are smoked inside residence (cig h^{-1} per smoker)
N_t	rate at which cigarettes are smoked inside a vehicle (cig h^{-1} per smoker)
PM	(airborne) particulate matter
$PM_{2.5}$	suspended particulate matter smaller than 2.5 micrometers in diameter
PM_{10}	suspended particulate matter smaller than 10 micrometers in diameter
PTEAM	particle total exposure assessment methodology study
Q	ventilation rate ($m^3\ h^{-1}$)
Q'(t)	time-dependent variation of ventilation rate relative to the mean ($m^3\ h^{-1}$)
Q_{avg}	time-averaged ventilation rate ($m^3\ h^{-1}$)
q_{avg}	time-averaged ventilation rate per building occupant, = Q_{avg}/Γ ($m^3\ h^{-1}$ person^{-1})
QL	quantifiable limit
R	fractional reduction in adult smoking prevalence between 1988 and 1998
RSP	respirable suspended particulate matter
RTI	Research Triangle Institute
S_i	ith value in a probability distribution function
SAS	Statistical Analysis Software

SD	standard deviation
SE	standard error
SRP	self-reported proximity (to environmental tobacco smoke)
SS	sidestream smoke; that which issues from the smoldering ember at the tip of a cigarette
t	time (h)
t_1	time at start of an exposure episode (h)
t_2	time at end of an exposure episode (h)
T	exposure period (h)
TAC	toxic air contaminants
TEAM	Total Exposure Assessment Methodology — monitoring studies designed to measure personal exposure to environmental contaminants, usually by multiple pathways
TH	exposure scenario based on ETS tracer measurements, using high-range parameters
TL	exposure scenario based on ETS tracer measurements, using low-range parameters
TM	exposure scenario based on ETS tracer measurements, using mid-range parameters
uncertainty	inaccuracies and imprecision resulting from imperfect information
variability	true differences in the value of a parameter when determined across a population
V	volume of microenvironment (m^3)
VOC	volatile organic compounds
X_i	mean ETS-only exposure concentration for compound i ($\mu g\ m^{-3}$)
X_r	mean ETS-only exposure concentration for reference compound r ($\mu g\ m^{-3}$)
x	exposure to a contaminant from sources other than ETS
y	exposure to a contaminant from ETS plus other sources
y^*	hypothetical exposure to a contaminant from ETS plus other sources
z	exposure to a contaminant from ETS only
Z	ETS exposure scale factor ($\mu g\ m^{-3}/(\mu g\ cig^{-1})$, or $cig\ m^{-3}$)
Γ	total number of occupants in a microenvironment
η	rate at which cigarettes are smoked in an environment (cigarettes per hour)
λ	air-exchange rate, = Q/V (h^{-1})
λ_{avg}	time-averaged air-exchange rate, = Q/V (h^{-1})

Appendix A. Concentrations of Indoor Air Toxicants from ETS

The research described in this report considers the hypothesis that ETS is a significant source of exposure to toxic air contaminants. The purpose of this appendix is to explore whether that hypothesis is supported by evidence from microenvironmental concentration measurements and models.

Initially, we conducted a descriptive analysis of the indoor air concentration measurements in the Californian Exposures Database (CED). In the absence of personal exposure measurements, indoor air concentrations are often used as surrogates to assess human exposures to indoor air contaminants. Results of the recently completed TEAM studies have shown that personal exposures are correlated with indoor air concentrations but that average exposure concentrations are typically higher than the corresponding indoor air concentrations (Hartwell et al., 1987; Pellizzari et al., 1987; Sheldon et al., 1992). Explaining why personal exposures are elevated above indoor concentrations is an active area of research. One determinant could be spatially varying concentrations in rooms containing point sources of emissions: concentrations tend to be greater in close proximity to the source than they are further from it (Miller-Leiden et al., 1996). Nevertheless, indoor air concentration measurements provide direct evidence concerning pollutant emissions into indoor air.

Qualitative insight into the contribution of ETS to concentrations of indoor air toxicants can be gleaned by comparing field measurements of indoor environments with and without smoking and to compare indoor with outdoor air concentrations. The levels of VOCs in smoking and nonsmoking indoor environments have been the focus of several field studies. Table A.1 summarizes the indoor with smoking (environments where active smoking occurred), indoor without smoking (environments where no smoking was reported), and outdoor air concentrations of selected air toxicants measured in a variety of settings, including the six studies in the CED. Where available, the arithmetic mean (AM) and range of concentrations are reported for the compounds included in the present study. (No such data were found for o-cresol, m,p-cresol, ethyl acrylate, or phenol.) A general trend can be seen from the table that applies to most compounds: indoor concentrations in smoking environments are higher than in nonsmoking environments, and indoor concentrations are higher than outdoor concentrations. It is noteworthy that this trend occurs across a variety of sites including residences (for example, in the TEAM studies), offices, and bars. These indoor environments have different ventilation rates and building volumes; the smoking rates (number of cigarettes smoked per hour) were also highly variable across studies. In addition, these studies were conducted in many different parts of the U.S., and in Britain. Overall, these observations suggest that smoking contributes significantly to

the indoor levels of air toxicants, but that sources other than tobacco smoke must be present as well.

In assessing the impact of ETS on personal exposure, it is important to consider the significance of other indoor sources. Many of the compounds have indoor sources in addition to ETS. For example, the indoor air concentration of benzene may have contributions from outdoor air (mostly motor vehicle emissions), gasoline vapor from attached garages, ETS, and a variety of building materials and consumer products. Resolving indoor air concentrations can be accomplished by estimating the contribution of each source separately. Material balance models can be applied to predict levels of air toxicants that result only from smoking.

We used the simplest form of a material balance model, the steady-state completely mixed room (CMR) model, to predict the magnitude of indoor air concentrations of air toxicants contributed by ETS. In this application, the model assumes that emissions are steady, that the indoor air is perfectly mixed, and that removal occurs only by ventilation. The model serves as a useful tool to estimate long-term average concentrations; its predictions can be compared with concentrations measured in field studies (National Research Council, 1986). With the steady-state CMR model, the indoor air concentration, C ($\mu g\ m^{-3}$), of a toxic air compound present in ETS is estimated to be

$$C = \frac{\eta\, e}{Q} \qquad (A.1)$$

where

η = rate at which cigarettes are smoked ($cig\ h^{-1}$)

e = emission factor for specific toxicant ($\mu g\ cig^{-1}$)

$Q = \lambda V$ = ventilation rate ($m^3\ h^{-1}$)

λ = air-exchange rate (h^{-1})

V = volume of indoor environment (m^3)

Key parameters for accurate estimation with the model are the emission factors and smoking rates. We used ETS emissions factors from Daisey et al. (1994, 1998) as inputs into equation (A.1) (see Table 3.1). Smoking rates were based on the 1994 update to the California Behavioral Risk Factor Survey (California Department of Health Services, 1995). This ongoing telephone survey assesses the prevalence of and trends in health-related behaviors in the adult Californian population. Respondents to the 1994 survey reported that 47–49% of California smokers consume 10 or fewer cigarettes per day, 36–40% smoke 11–20 cigarettes per day, and 11–17% smoke more than 20 cigarettes per day. For our predictions, smoking frequencies corresponding roughly to

those of a single light smoker (10 cigarettes per day) and moderately heavy smoker (30 cigarettes per day) were used.

We modeled an average and worst-case residential ventilation scenario. These scenarios incorporate data from the U.S. distribution of residential air exchange rates, broken down by region and by season (Murray and Burmaster, 1995). For California, the average air exchange rates for all seasons are 0.55 and 0.98 h^{-1} in Northern and Southern California, respectively. The 10th percentile air-exchange rates for these 2 regions are 0.20 and 0.26 h^{-1}, respectively. Therefore, rates of 0.5 and 0.2 h^{-1} were selected to represent typical and worst-case scenarios. Finally, a home volume of 300 m^3, the median volume of a three-person house (Residential Energy Consumption Survey, 1982), was used for our predictions.

Figure A.1 compares the predictions from equation (A.1) to the average excess air toxicant concentrations indoors due to ETS for the six studies in the CED. To appropriately include the CED data in comparison, we removed the effects of other sources of air toxicants beside ETS. We calculated the measured concentration of an air toxicant due to ETS alone as follows: (a) the outdoor levels were subtracted from the indoor concentrations for both the smoking and nonsmoking homes (for Study 1, the outdoor air concentrations were greater than the nonsmoking indoor air concentrations); (b) the results for smoking and nonsmoking homes were then averaged across the six studies; and (c) the difference between the smoking and nonsmoking homes was plotted in Figure A.1. In general, the CMR model predicts well the excess indoor concentrations of air toxicants from ETS, except for styrene. Overall, this concordance is encouraging, because it substantiates the hypothesis that the exposures to specific TACs in ETS scale with the emission factors. The results presented in Figure A.1 also support the hypothesis that ETS is an important indoor source of TACs.

Table A.1. Field measurements of air toxicants in ETS: Concentrations ($\mu g\ m^{-3}$) in smoking and nonsmoking indoor environments and in outdoor air.

	smoking		*nonsmoking*		*outdoors*		
compound	AM	range[1]	AM	range[1]	AM	range[1]	comments
acetaldehyde		183-204				ND	tavern[2]
	462	170-630					5 cafes[3]
acrylonitrile		1.8					bowling alley[4]
		0.8		ND			residence, smkg room[4]
		0.6		ND			residence, remote from smkg room[4]
	1.9						restaurant[4]
benzene	21	2.4-43	14	0.73-34	16	4.3-28	Study 1, LA county, CA (winter '84)[5]
	7.8	0.02-25	8.4	0.66-35	3.6	1.2-8.7	Study 2, LA county, CA (summer '84)[5]
	8.5	0.14-24	4.6	0.91-17	1.8	0.79-3.8	Study 3, Pittsburg/Antioch, CA (summer '84)[5]
	14	2.2-40	12	1.5-61	8.2	0.86-20	Study 4, LA county, CA (winter '87)[5]
	9.1	1.5-36	5.2	1.6-17	4.0	0.87-11	Study 5, LA county, CA (summer '87)[5]
	8.4	0.33-130	2.7	0-9.4	1.2	0.46-3.0	Study 6, Woodland, CA (summer '90)[5]
	16		8.4		8.6		TEAM study, New Jersey (fall '81)[6]
	96	50-150					5 cafes[3]
		ND-18.3		ND-10.8			3 office complexes[7]
		21-27				6-8	tavern[2]
		10.2					bowling alley[4]
		17.6		3.6			residence, smoking room[4]
		6.9		2.8			residence, remote from smkg room[4]
	12.4						restaurant[4]
	13	3-49	12	3-31			7 smkg, 3 nonsmkg offices[8]
	17	9-30	9.5	9-10			5 smkg, 1 nonsmkg betting shop[9]

Table A.1. (continued)

	smoking		*nonsmoking*		*outdoors*		
compound	AM	range[1]	AM	range[1]	AM	range[1]	comments
1,3-butadiene		11-19				<1-1	tavern[2]
2-butanone (MEK)		17.7					bowling alley[4]
		18.5		9.5			residence, smkg room[4]
		12.4		6.8			residence, remote from smkg room[4]
	20.4						restaurant[4]
ethylbenzene	11	1.0-23	7.5	1.1-29	9.8	0.56-19	Study 1, LA county, CA (winter '84)[5]
	7.5	0.03-23	6.2	0.14-35	3.6	0.75-17	Study 2, LA county, CA (summer '84)[5]
	3.0	0.32-10	2.4	0.17-9.5	1.1	0.12-4.0	Study 3, Pittsburg/Antioch, CA (summer '84)[5]
	4.9	2.0-7.9	5.0	0.93-28	3.4	0.31-9.4	Study 4, LA county, CA (winter '87)[5]
	2.9	1.7-4.4	2.8	0.7-13	1.8	0.34-6.3	Study 5, LA county, CA (summer '87)[5]
	7.9		4.5		3.8		TEAM study, New Jersey (fall '81)[6]
	3.9		4.8		3.5		TEAM study, New Jersey (summer '82)[6]
	5.6		7.5		3.4		TEAM study, New Jersey (winter '83)[6]
		ND-0.04		ND-22			3 office complexes[6]
		22.2					bowling alley[4]
		8.0		3.9			residence, smkg room[4]
		2.5		3.1			residence, remote from smkg room[4]
	6.2						restaurant[4]
	12	2-122	5	1-13			7 smkg, 3 nonsmkg offices[8]
	14.5	9-32	11	11-11			5 smkg, 1 nonsmkg betting shop[9]
formaldehyde	85.9		110				26 nonsmkg homes, 17 smkg homes[10]
		89-104				ND	tavern[2]

Table A.1. (continued)

compound	*smoking* AM	*smoking* range[1]	*nonsmoking* AM	*nonsmoking* range[1]	*outdoors* AM	*outdoors* range[1]	comments
N-nitroso-dimethylamine	0.03	ND-0.05					4 restaurants[11]
		0.07					bar[11]
	0.03	0.02-0.033					4 offices[11]
styrene	4.8	0.03-9.2	2.7	0.02-8.9	3.8	0.64-9.1	Study 1, LA county, CA (winter '84)[5]
	1.73	0.02-5.3	0.94	0.02-3.2	0.68	0.21-2.2	Study 2, LA county, CA (summer '84)[5]
	1.2	0.23-4.1	0.7	0.02-2.9	0.54	0.02-1.4	Study 3, Pittsburg/Antioch, CA (summer '84)[5]
	3.2	1.1-5.1	1.9	0.55-4.5	1.7	0.04-6.1	Study 4, LA county, CA (winter '87)[5]
	1.3	0.35-3.2	0.98	0.25-4.3	0.46	0.05-1.9	Study 5, LA county, CA (summer '87)[5]
	1.9	0.11-14	6.3	0-135	0.24	0-1.9	Study 6, Woodland, CA (summer '90)[5]
	1.9		1.0		0.9		TEAM study, New Jersey (fall '81)[6]
	1.4		1.2		0.6		TEAM study, New Jersey (summer '82)[6]
	1.5		1.0		0.6		TEAM study, New Jersey (winter '83)[6]
		185					bowling alley[4]
		7.3		2.0			residence, smkg room[4]
		3.0		1.6			residence, remote from smkg room[4]
	4.4						restaurant[4]
	14	2-59	18	4-79			7 smkg, 3 nonsmkg offices[8]
	6.8	4-11	5	4-6			5 smkg, 1 nonsmkg betting shop[9]
toluene	545	40-1040					4 cafes[3]
		0.6-248		0.9-142			3 office complexes[7]
		40.2					bowling alley[4]
		51.2		20.9			residence, smkg room[4]
		25.0		16.0			residence, remote from smkg room[4]
	77.3						restaurant[4]
	40	10-292	26	7-65			7 smkg, 3 nonsmkg offices[8]
	59.6	28-120	37	35-39			5 smkg, 1 nonsmkg betting shop[9]

Table A.1. (continued)

compound	*smoking* AM	*smoking* range[1]	*nonsmoking* AM	*nonsmoking* range[1]	*outdoors* AM	*outdoors* range[1]	comments
o-xylene	12.1	1.6-22	9.8	1.2-34	11	2.2-21	Study 1, LA county, CA (winter '84)[5]
	8.0	0.03-25	5.9	0.02-34	3.0	0.77-8.8	Study 2, LA county, CA (summer '84)[5]
	3.5	0.42-12	3.2	0.28-13	0.83	0.07-2.9	Study 3, Pittsburg/Antioch, CA (summer '84)[5]
	9.5	3.7-16	9.6	2.0-48	7.0	0.64-20	Study 4, LA county, CA (winter '87)[5]
	4.3	3.2-6.8	4.7	1.2-18	2.9	0.43-10	Study 5, LA county, CA, CA (summer '87)[5]
	2.9	0.22-14	2.5	0-9.85	0.89	0.26-2.3	Study 6, Woodland, CA, CA (summer '90)[5]
	6.3		3.8		4.0		TEAM study, New Jersey (fall '81)[6]
	4.6		5.5		4.3		TEAM study, New Jersey (summer '82)[6]
	6.1		7.2		3.1		TEAM study, New Jersey (winter '83)[6]
		0.08-78		ND-9.2			3 office complexes[7]
		15.2					bowling alley[4]
		7.1		5.4			residence, smkg room[4]
		4.9		3.7			residence, remote from smkg room[4]
	6.8						restaurant[4]
	14	3-68	12	5-27			7 smkg, 3 nonsmkg offices[8]
	12.4	6-25	11	11-11			5 smkg, 1 nonsmkg betting shop[9]
m,p-xylene	27	4.7-51	21	4.1-58	25	6.4-51	Study 1, LA county, CA (winter '84)[5]
	27	0.60-92	18	1.2-94	11	3.1-50	Study 2, LA county, CA (summer '84)[6]
	9.3	1.2-29	8.3	1.0-30	2.8	0.38-11	Study 3, Pittsburg/Antioch, CA (summer '84)[5]
	26	9.4-46	26	5.7-126	19	1.65-50	Study 4, LA county, CA (winter '87)[5]
	13	8.3-20	12	3.0-52	8.2	1.3-26	Study 5, LA county, CA (summer '87)[5]
	6.5	0.47-29	5.1	0-20	1.7	0.48-4.3	Study 6, Woodland, CA (summer '90)[5]

Table A.1. (continued)

compound	*smoking* AM	*smoking* range[1]	*nonsmoking* AM	*nonsmoking* range[1]	*outdoors* AM	*outdoors* range[1]	comments
m,p-xylene (cont'd)	19		11		11		TEAM study, New Jersey (fall '81)[6]
	11		13		11		TEAM study, New Jersey (summer '82)[6]
	17		21		8.5		TEAM study, New Jersey (winter '83)[6]
		659					Bowling alley[4]
		22.4		11.3			residence, smkg room[4]
		12.0		7.7			residence, remote from smkg room[4]
	13.2	21.4					restaurant[4]
	73	14-328	70	23-170			7 smkg, 3 nonsmkg offices[8]
	35.4	16-77	27	27-27			5 smkg, 1 nonsmkg betting shop[9]

[1] A single number indicates only one measurement reported.
[2] Löfroth et al. (1989).
[3] Badre et al. (1978).
[4] Guerin (1996); Guerin et al. (1992).
[5] Californian Exposures Database; for Studies 1-3, overnight personal air samples were used to represent indoor air concentrations; for Studies 4-6, indoor air concentrations were sampled in main living area.; smoking measurements are from the active subpopulation; nonsmoking measurements are from the unexposed subpopulation.
[6] Wallace et al. (1987); Wallace (1987); weighted GMs of overnight personal air samples were used to represent indoor air concentrations; weighted AM of outdoor concentrations is reported.
[7] Bayer and Black (1987).
[8] Proctor et al. (1989a).
[9] Proctor et al. (1989b).
[10] Stock (1987).
[11] Stehlik et al. (1982).
AM = arithmetic mean.
ND = not detected.

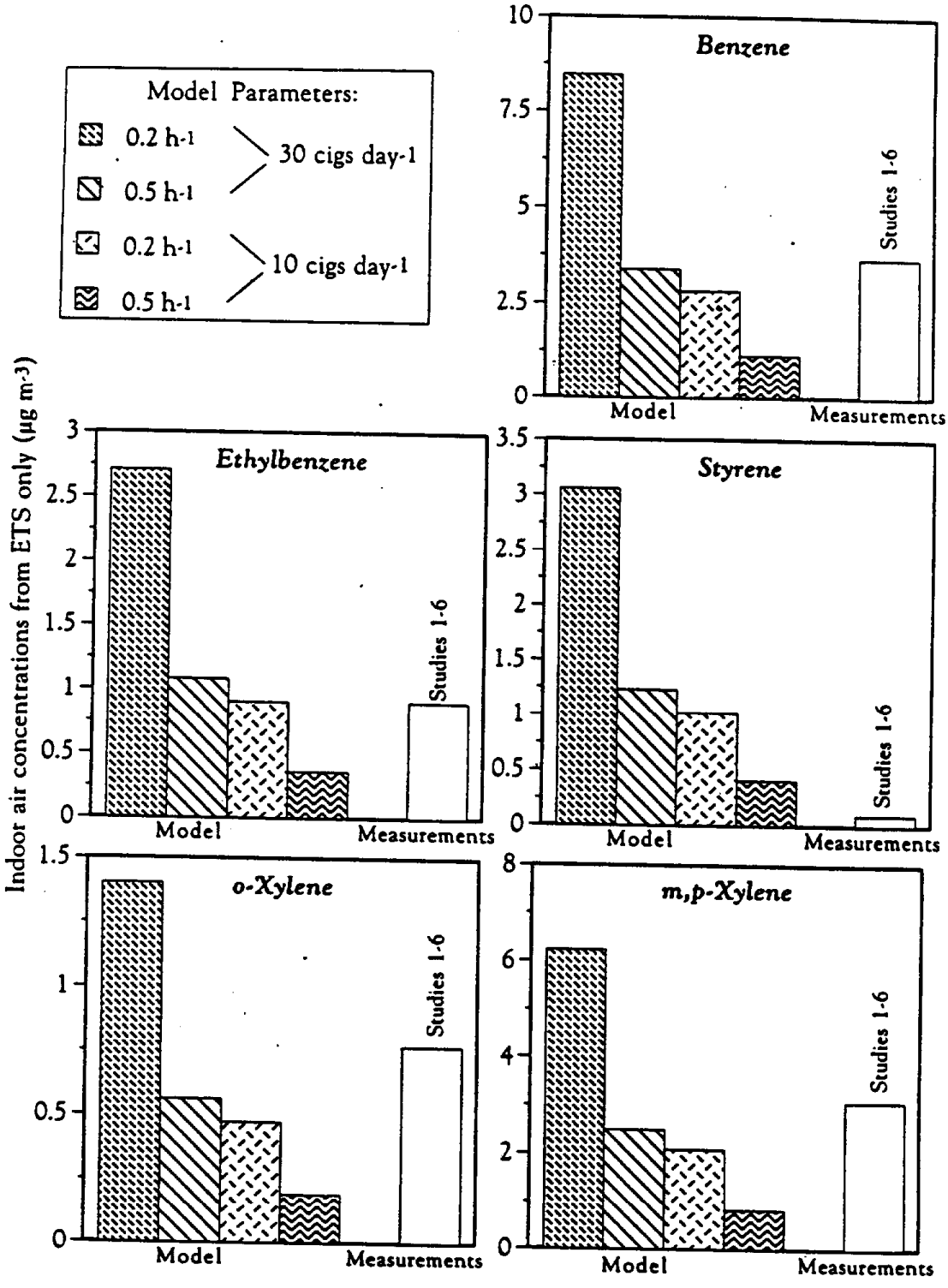

Figure A.1 Excess indoor air concentrations of TAC associated with ETS as measured in homes compared to predictions from a completely mixed room model.

Appendix B. Daily Personal Exposures to Air Toxicants

We conducted many analyses using the 24-hour personal exposure data from the CED to better understand the characteristics of personal exposures to air toxicants, the factors influencing exposure, and the sources of exposure. These analyses focus on the five ETS-related compounds that are most commonly above the measurement detection limit: benzene, ethylbenzene, styrene, o-xylene, and m,p-xylene.

Table B.1 presents the arithmetic mean (AM) and range of 24-hour personal exposures for both active smokers and persons unexposed to ETS for the six CED studies, and for four other studies of personal exposures to air toxicants: three New Jersey TEAM studies and a study by Proctor et al. (1991). The study by Proctor and coworkers assessed 24-hour personal VOC exposures of fifty-two smoking and nonsmoking British women in the autumn of 1989 using questionnaires, personal monitoring, and analysis of cotinine in saliva. Generally, the average personal exposure levels are similar among all 10 studies, spanning less than an order of magnitude for all compounds except styrene; styrene arithmetic means range from 1.3 to 18 $\mu g\ m^{-3}$ for smokers. In most cases, the nonsmoking exposures are less than the smoking exposures; by contrast, the nonsmoking average exposure is higher than the smoking exposure for Studies 5 and 6 for some compounds, most likely due to the large variability in the measurements and the small sample sizes. Overall, these results, which are similar to the field measurements of indoor air concentrations, give strong evidence that cigarette smoke is an important source of some air toxicants. The data also suggest that exposures are not highly variable across diverse study populations.

B.1 Univariate Statistics

An important step in our research was to construct a descriptive summary of the data in the CED that highlighted its salient features for exploring TAC exposure due to ETS. We computed descriptive statistics for three exposure subpopulations: active, passive, and unexposed. Tables B.2–B.7 summarize 24-hour personal exposure measurements for each of the six studies in the CED. The statistical parameters we report include the range of exposure levels (minimum and maximum), sample population arithmetic mean (AM), standard deviation (SD), geometric mean (GM), geometric standard deviation (GSD), 25, 50, and 75th percentiles, standard error of the sample mean (SE), and coefficient of variation (COV). We computed weighted statistics for the data collected in Studies 1, 2, 3, and 6. Participants in these studies were selected using probability-based sampling. We used the inverse of their selection probabilities as weights in order to appropriately account for the sampling design structure. We applied unweighted procedures for Studies 4 and 5.

Table B.1. Field measurements of air toxicants in ETS: Smokers and nonsmokers 24-hour personal exposure concentrations ($\mu g\ m^{-3}$).

	smokers		*nonsmokers*		
compound	AM	range	AM	range	comments
benzene	21.6	5.2-103	13.2	0.2-32.1	11 smkg women; 17 nonsmkg women[1]
	22	5.5-48	16	4.0-45	Study 1, LA county, CA (winter '84)[2]
	8.3	1.1-25	8.0	1.4-22	Study 2, LA county CA (summer '84)[2]
	9.1	1.8-23	6.5	1.7-20	Study 3, Pittsburg/Antioch, CA (summer '84)[2]
	24	13-61	15	2.4-67	Study 4, LA county, CA (winter '87)[2]
	7.9	3.4-20	10	1.6-64	Study 5, LA county, CA (summer '87)[2]
	5.6	1.5-46	4.9	0.35-42	Study 6, Woodland, CA (summer '90)[2]
	18		11		TEAM study, New Jersey (fall '81)[3]
ethylbenzene	10.1	4.0-14.6	11.1	0.3-52.1	11 smkg women; 17 nonsmkg women[1]
	15	4.5-68	9.4	1.3-51	Study 1, LA county, CA (winter '84)[2]
	9.0	0.07-40	6.0	0.67-24	Study 2, LA county CA (summer '84)[2]
	3.8	0.92-12	3.7	0.59-17	Study 3, Pittsburg/Antioch, CA (summer '84)[2]
	8.4	4.4-11	7.1	0.77-29	Study 4, LA county, CA (winter '87)[2]
	3.2	1.5-5.6	7.0	0.82-63	Study 5, LA county, CA (summer '87)[2]
	10		8		TEAM study, New Jersey (fall '81)[3]
	4		4		TEAM study, New Jersey (summer '82)[3]
	11		8		TEAM study, New Jersey (winter '82)[3]
styrene	3.0	1.0-9.7	2.9	0.8-12.4	11 smkg women; 17 nonsmkg women[1]
	4.8	1.1-13	2.9	0.07-11	Study 1, LA county, CA (winter '84)[2]
	2.5	0.21-5.9	1.0	0.02-3.0	Study 2, LA county CA (summer '84)[2]
	1.3	0.43-5.6	0.79	0.14-2.5	Study 3, Pittsburg/Antioch, CA (summer '84)[2]
	18	2.2-163	2.2	0.22-4.7	Study 4, LA county, CA (winter '87)[2]
	1.8	0.19-3.0	3.8	0.14-37	Study 5, LA county, CA (summer '87)[2]
	1.7	0.3-3.7	3.3	0.2-48	Study 6, Woodland, CA (summer '90)[2]
	2.6		1.8		TEAM study, New Jersey (fall '81)[3]
	1.7		1.1		TEAM study, New Jersey (summer '82)[3]
	2.7		1.5		TEAM study, New Jersey (winter '82)[3]

Table B.1. (continued)

	smoking		*nonsmoking*		
compound	AM	range	AM	range	comments
o-xylene	10.2	3.8-16.4	9.5	1.0-17.0	11 smkg women; 17 nonsmkg women[1]
	5	5.1-42	11	2.0-48	Study 1, LA county, CA (winter '84)[2]
	7.6	0.03-33	5.6	0.81-20	Study 2, LA county CA (summer '84)[2]
	4.1	0.83-12	4.3	0.72-16	Study 3, Pittsburg/Antioch, CA (summer '84)[2]
	19	6.7-75	14	1.3-63	Study 4, LA county, CA (winter '87)[2]
	4.5	2.1-7.7	10	1.2-83	Study 5, LA county, CA (summer '87)[2]
	3.3	1.0-8.9	5.2	0.5-44	Study 6, Woodland, CA (summer '90)[2]
	8		7		TEAM study, New Jersey (fall '81)[3]
	5		5		TEAM study, New Jersey (summer '82)[3]
	13		9		TEAM study, New Jersey (winter '82)[3]
m,p-xylene	30.6	4.7-102	34.0	3.4-166	11 smkg women; 17 nonsmkg women[1]
	33	13-118	25	6.4-77	Study 1, LA county, CA (winter '84)[2]
	29	0.58-90	18	3.5-71	Study 2, LA county CA (summer '84)[2]
	12	3.3-28	11	1.9-37	Study 3, Pittsburg/Antioch, CA (summer '84)[2]
	53	24-216	35	3.8-142	Study 4, LA county, CA (winter '87)[2]
	13	6.6-24	27	3.4-217	Study 5, LA county, CA (summer '87)[2]
	7.0	1.8-18	11	1.3-84	Study 6, Woodland, CA (summer '90)[2]
	25		19		TEAM study, New Jersey (fall '81)[3]
	12		10		TEAM study, New Jersey (summer '82)[3]
	33		23		TEAM study, New Jersey (winter '82)[3]

[1] Proctor et al. (1991).
[2] Californian Exposures Database; smokers are participants who reported actively smoking and nonsmokers are those who reported being unexposed to ETS during study.
[3] Wallace et al. (1987); weighted GMs of daytime personal air samples are reported.
AM = arithmetic mean.

Table B.2. Univariate statistics for 24-hour personal exposures, Study 1, LA County, winter 1984.

compound	*exposure category*	*# obs*	*range* ($\mu g\ m^{-3}$)	*AM* ($\mu g\ m^{-3}$)	*SD* ($\mu g\ m^{-3}$)	*GM* ($\mu g\ m^{-3}$)	*GSD*	*25th %ile* ($\mu g\ m^{-3}$)	*50th %ile* ($\mu g\ m^{-3}$)	*75th %ile* ($\mu g\ m^{-3}$)	*SE* ($\mu g\ m^{-3}$)	*COV*
benzene	active	34	5.5-48	22	9.9	20	1.6	14	19	29	1.7	45%
	passive	23	4.8-27	16	6.2	15	1.6	11	16	19	1.3	39%
	unexposed	53	4.0-45	16	8.8	13	1.8	9.7	14	19	1.2	55%
styrene	active	34	1.1-13	4.8	2.4	4.3	1.6	3.0	4.7	6.0	0.41	50%
	passive	22	0.46-11	3.2	2.2	2.5	2.0	1.9	2.9	3.9	0.47	69%
	unexposed	53	0.07-11	2.9	2.2	2.2	2.5	1.7	2.3	3.3	0.31	76%
ethylbenzene	active	34	4.5-68	15	11	12	1.7	8.4	11	16	1.9	73%
	passive	22	2.5-30	9.9	6.3	8.3	1.8	6.8	7.7	12	1.3	64%
	unexposed	53	1.3-51	9.4	8.7	7.1	2.0	4.8	7.5	11	1.2	93%
o-xylene	active	34	5.1-42	15	8.0	13	1.6	10	12	17	1.4	53%
	passive	22	3.2-34	13	7.1	11	1.8	7.7	11	15	1.5	55%
	unexposed	53	2.0-48	11	8.0	9.4	1.9	6.8	10	14	1.1	73%
m,p-xylene	active	34	13-118	33	18	30	1.5	23	29	38	3.1	55%
	passive	22	7.2-60	26	13	24	1.6	17	22	35	2.7	50%
	unexposed	53	6.4-77	25	14	21	1.7	18	21	34	2.0	56%

Table B.3. Univariate statistics for 24-hour personal exposures, Study 2, LA County, summer 1984.

compound	*exposure category*	*# obs*	*range* ($\mu g\ m^{-3}$)	*AM* ($\mu g\ m^{-3}$)	*SD* ($\mu g\ m^{-3}$)	*GM* ($\mu g\ m^{-3}$)	*GSD*	*25th %ile* ($\mu g\ m^{-3}$)	*50th %ile* ($\mu g\ m^{-3}$)	*75th %ile* ($\mu g\ m^{-3}$)	*SE* ($\mu g\ m^{-3}$)	*COV*
benzene	active	16	1.1-25	8.3	5.8	6.6	2.0	4.1	7.3	11	1.5	70%
	passive	14	1.8-26	11	8.1	8.2	2.3	4.6	9.3	17	2.2	74%
	unexposed	20	1.4-22	8.0	6.2	5.8	2.3	2.8	5.4	11	1.4	78%
styrene	active	16	0.21-5.9	2.5	1.8	1.8	2.7	0.87	1.5	3.7	0.44	72%
	passive	14	0.03-6.5	2.0	1.7	1.3	3.4	1.0	1.8	3.0	0.44	85%
	unexposed	20	0.02-3.0	1.0	0.79	0.62	3.7	0.41	0.85	1.7	0.18	79%
ethylbenzene	active	16	0.07-40	9.0	7.2	6.5	2.6	3.6	6.4	12	1.8	80%
	passive	14	0.13-23	8.0	6.7	5.0	3.4	3.5	6.4	11	1.8	84%
	unexposed	20	0.67-24	6.0	6.3	3.9	2.5	1.8	2.8	7.0	1.4	105%
o-xylene	active	16	0.03-33	7.6	7.4	4.2	3.8	2.8	5.1	11	1.9	97%
	passive	14	0.25-31	9.0	8.0	5.8	3.0	4.3	6.8	10	2.1	89%
	unexposed	20	0.81-20	5.6	6.2	3.3	2.7	1.4	2.3	6.7	1.4	111%
m,p-xylene	active	16	0.58-90	29	20	22	2.4	12	20	42	4.9	69%
	passive	14	0.87-76	28	22	18	2.9	13	21	44	5.8	79%
	unexposed	20	3.5-71	18	18	12	2.3	6.1	9.9	16	4.1	100%

Table B.4. Univariate statistics for 24-hour personal exposures, Study 3, Pittsburg/Antioch, summer 1984.

compound	*exposure category*	*# obs*	*range* ($\mu g\ m^{-3}$)	*AM* ($\mu g\ m^{-3}$)	*SD* ($\mu g\ m^{-3}$)	*GM* ($\mu g\ m^{-3}$)	*GSD*	*25th %ile* ($\mu g\ m^{-3}$)	*50th %ile* ($\mu g\ m^{-3}$)	*75th %ile* ($\mu g\ m^{-3}$)	*SE* ($\mu g\ m^{-3}$)	*COV*
benzene	active	20	1.8-22	9.1	5.5	7.6	1.8	4.1	6.2	12	1.2	60%
	passive	18	2.5-25	7.4	5.5	6.0	1.8	3.7	5.7	9.1	1.3	74%
	unexposed	29	1.7-20	6.5	4.4	5.4	1.8	4.1	5.6	10	0.82	68%
styrene	active	20	0.43-5.6	1.3	0.95	1.1	1.8	0.72	1.0	1.7	0.21	73%
	passive	18	0.18-2.0	0.90	0.60	0.69	2.2	0.45	0.82	1.2	0.14	67%
	unexposed	28	0.14-2.5	0.79	0.51	0.64	2.0	0.47	0.77	1.3	0.10	65%
ethylbenzene	active	20	0.92-12	3.8	3.0	2.9	2.1	1.6	2.8	5.3	0.67	79%
	passive	18	0.78-21	3.6	4.8	2.3	2.4	1.1	2.3	4.1	1.1	133%
	unexposed	29	0.59-17	3.7	3.2	2.8	2.0	2.1	2.9	5.0	0.59	86%
o-xylene	active	20	0.83-12	4.1	2.7	3.3	2.0	2.2	3.1	5.2	0.6	66%
	passive	18	1.1-26	4.6	6.1	2.9	2.3	1.5	2.8	5.6	1.4	133%
	unexposed	29	0.72-16	4.3	3.4	3.4	2.0	2.6	3.9	6.2	0.64	79%
m,p-xylene	active	20	3.3-28	12	7.2	9.5	1.9	6.2	10	14	1.6	60%
	passive	18	3.3-53	11	12	7.9	2.1	4.3	7.3	14	2.8	109%
	unexposed	29	1.9-37	11	7.8	8.7	2.0	6.8	9.6	15	1.4	71%

Table B.5. Univariate statistics for 24-hour personal exposures, Study 4, LA County, winter 1987.

compound	*exposure category*	*# obs*	*range* ($\mu g\ m^{-3}$)	*AM* ($\mu g\ m^{-3}$)	*SD* ($\mu g\ m^{-3}$)	*GM* ($\mu g\ m^{-3}$)	*GSD*	*25th %ile* ($\mu g\ m^{-3}$)	*50th %ile* ($\mu g\ m^{-3}$)	*75th %ile* ($\mu g\ m^{-3}$)	*SE* ($\mu g\ m^{-3}$)	*COV*
benzene	active	8	13-61	24	15	21	1.6	15	19	27	5.4	63%
	passive	9	5.0-23	13	5.5	12	1.7	12	14	15	1.8	42%
	unexposed	12	2.4-67	15	16	11	2.2	7.3	9.9	17	4.8	107%
styrene	active	12	2.2-163	18	44	5.7	3.0	2.9	5.2	6.6	13	244%
	passive	11	0.96-7.5	3.6	2.2	3.0	1.9	1.6	2.7	5.9	0.67	61%
	unexposed	16	0.22-4.7	2.2	1.3	1.7	2.1	1.1	1.9	3.2	0.32	59%
ethylbenzene	active	11	4.4-11	8.4	2.0	8.1	1.3	6.7	8.3	11	0.61	24%
	passive	11	2.7-18	8.3	5.1	6.8	1.9	3.5	6.1	13	1.5	61%
	unexposed	16	0.77-29	7.1	8.4	4.5	2.4	2.5	4.4	6.1	2.1	118%
o-xylene	active	12	6.7-75	19	17	15	1.7	11	15	17	5.0	89%
	passive	11	4.8-41	17	12	13	2.1	6.4	13	27	3.6	71%
	unexposed	16	1.3-63	14	16	8.7	2.6	4.2	8.5	13	4.1	114%
m,p-xylene	active	12	24-216	53	50	44	1.7	33	40	46	14	94%
	passive	11	12-93	43	27	35	1.9	18	34	62	8.1	63%
	unexposed	16	3.8-142	35	37	23	2.4	12	23	33	9.3	106%

Table B.6. Univariate statistics for 24-hour personal exposures, Study 5, LA County, summer 1987.

compound	*exposure category*	*# obs*	*range* ($\mu g\ m^{-3}$)	*AM* ($\mu g\ m^{-3}$)	*SD* ($\mu g\ m^{-3}$)	*GM* ($\mu g\ m^{-3}$)	*GSD*	*25th %ile* ($\mu g\ m^{-3}$)	*50th %ile* ($\mu g\ m^{-3}$)	*75th %ile* ($\mu g\ m^{-3}$)	*SE* ($\mu g\ m^{-3}$)	*COV*
benzene	active	11	3.4-20	7.9	4.3	7.1	1.6	5.2	7.1	8.7	1.3	54%
	passive	12	3.0-53	12	13	8.6	2.1	5.1	6.0	14	3.8	108%
	unexposed	15	1.6-64	10	15	6.7	2.2	4.8	6.7	8.5	3.8	150%
styrene	active	11	0.19-3.0	1.8	0.83	1.4	2.2	1.0	1.7	2.5	0.25	46%
	passive	10	0.50-3.8	1.6	9.7	1.3	1.9	0.73	1.6	2.0	0.31	63%
	unexposed	13	0.14-37	3.8	10	1.0	3.6	0.73	0.85	1.0	2.7	255%
ethylbenzene	active	11	1.5-5.6	3.2	1.2	2.9	1.5	2.0	3.1	4.2	0.35	38%
	passive	12	2.0-27	5.1	6.8	3.6	2.0	2.4	2.9	4.2	2.0	133%
	unexposed	15	0.82-63	7.0	15	3.2	2.7	1.6	2.8	4.2	3.9	214%
o-xylene	active	11	2.1-7.7	4.5	1.6	4.2	1.5	3.5	4.6	5.6	0.47	36%
	passive	12	1.6-38	7.2	9.5	4.9	2.1	3.1	4.4	6.1	2.7	132%
	unexposed	15	1.2-83	10	20	5.0	2.7	2.3	4.8	7.0	5.1	200%
m,p-xylene	active	11	6.5-24	13	4..9	12	1.4	9.1	13	18	1.5	38%
	passive	12	4.5-100	20	24	14	2.0	9.9	14	17	7.1	120%
	unexposed	15	3.4-217	27	51	14	2.6	6.3	13	20	13	189%

Table B.7. Univariate statistics for 24-hour personal exposures, Study 6, Woodland, summer 1990.

compound	*exposure category*	*# obs*	*range* ($\mu g\ m^{-3}$)	*AM* ($\mu g\ m^{-3}$)	*SD* ($\mu g\ m^{-3}$)	*GM* ($\mu g\ m^{-3}$)	*GSD*	*25th %ile* ($\mu g\ m^{-3}$)	*50th %ile* ($\mu g\ m^{-3}$)	*75th %ile* ($\mu g\ m^{-3}$)	*SE* ($\mu g\ m^{-3}$)	*COV*
benzene	active	21	1.5-46	5.7	8.4	3.9	2.0	2.6	3.3	5.4	1.8	147%
	passive	28	1.20-18	4.8	4.0	3.6	2.0	1.9	2.9	6.1	0.75	83%
	unexposed	44	0.35-42	4.9	7.2	3.0	2.5	1.8	3.0	5.3	1.1	147%
styrene	active	21	0.31-3.7	1.7	1.0	1.4	2.0	0.84	1.5	2.0	0.22	59%
	passive	28	0.23-6.3	1.6	1.4	1.1	2.3	0.68	1.0	2.2	0.26	88%
	unexposed	44	0.20-48	3.3	9.0	1.3	2.9	0.72	1.2	1.9	1.4	273%
ethylbenzene	active	not measured										
	passive											
	unexposed											
o-xylene	active	21	1.0-8.9	3.3	2.0	2.8	1.8	1.6	3.1	3.8	0.45	61%
	passive	28	0.37-24	4.5	5.7	2.7	2.7	2.0	2.7	4.3	1.1	127%
	unexposed	44	0.50-44	5.2	6.9	3.3	2.5	2.0	2.9	4.9	1.0	133%
m,p-xylene	active	21	1.8-18	7.0	4.7	5.8	1.9	3.0	6.4	8.1	1.0	67%
	passive	28	0.79-44	9.2	11	5.7	2.6	3.9	6.1	9.0	2.0	120%
	unexposed	44	1.3-84	11	13	6.7	2.5	4.0	5.9	11	2.0	118%

Table B.8. Yearly and seasonal relationships among 24-hour personal exposures: Pearson and Spearman rank correlation coefficients for comparisons between studies.

	yearly (1984 vs. 1987)						*seasonal (winter vs. summer)*					
	Study 1 vs. Study 4			Study 1 vs. Study 5			Study 1 vs. Study 2			Study 4 vs. Study 5		
compound	# obs[1]	Pearson	Spearman	# obs[1]	Pearson	Spearman	# obs[1]	Pearson	Spearman	# obs[1]	Pearson	Spearman
benzene	19	0.48*	0.64*	20	0.37	0.24	49	0.15	0.19	21	0.38	0.32
ethylbenzene	22	0.38	0.61*	19	0.43	0.45	48	0.08	0.26	28	0.42*	0.49*
styrene	23	0.12	0.37	18	0.42	0.58*	48	0.48*	0.35*	26	-0.06	0.44*
o-xylene	23	0.39	0.63*	19	0.48*	0.59*	48	0.14	0.10	29	0.37	0.53*
m,p-xylene	23	0.26	0.65*	19	0.39	0.46*	48	0.12	0.16	29	0.29	0.57*

[1]Number of observations compared in correlation analysis.

* indicates coefficient significantly different from zero at $p < 0.05$.

This type of analysis has been done by other researchers for some of these data. Our results agree exactly with the descriptive statistics for the ETS exposure categories for benzene and styrene for Studies 1 and 6 that are presented in Clayton and Perritt (1993). We are also in close agreement with the results of Wallace et al. (1987): they reported unweighted GMs for 12-hour daytime personal benzene exposures measured in Study 1 for smokers and nonsmokers to be 18 $\mu g\ m^{-3}$ (29 subjects) and 14 $\mu g\ m^{-3}$ (85 subjects), respectively. Our results in Table B.2 list the weighted geometric 24-hour personal benzene exposure as 20 $\mu g\ m^{-3}$ (34 subjects) for smokers and 13 $\mu g\ m^{-3}$ (53 subjects) for unexposed, respectively. The slight discrepancy between our results and those of Wallace et al. (1987) is most likely due to the different exposure classification methods and 12-hour versus 24-hour measurement periods.

Studies 5 and 6 each showed several compounds for which the unexposed mean exposure was higher than the mean for active smokers. The exposure variability of these compounds was also very high. The coefficient of variation (COV), which expresses the dispersion of exposures on a relative basis, indicates that the higher mean for the unexposed group may be a statistical aberration resulting from the large variance in the data. For Study 5, the COV for benzene, ethylbenzene, styrene, o-xylene and m,p-xylene ranged from 150 to 270%. By comparison, the COV for these same compounds in Study 1 was in the range 60-90%. Studies 2 and 3 each showed a passive mean exposure that was higher than the mean for active smokers for one or two compounds (benzene, o-xylene). The standard error of the sample mean (SE) was higher for the passive than the active population, indicating higher uncertainty. We suspect that the large variability in measurements and the small sample sizes account for these seemingly contradictory exposure levels of the different ETS exposure subpopulations.

B.2 Correlation and Frequency Analyses

To further explore the factors influencing exposure to TACs, we conducted a suite of correlation and frequency analyses using the exposures and participant/household characteristics contained in the CED. Each analysis was designed to investigate potential relationships that may exist between exposures to the compounds of interest and the presence of tobacco smoke; to determine whether these compounds are emitted from similar sources; and to identify which participant/household characteristics significantly influence exposure levels. We also investigated correlations over time of exposures to a compound, in order to gain insight into the temporal fluctuations of exposures.

To reduce the impact of sample sizes on some of our analyses, we created a data set that combined observations from all six studies, for a total sample size of 462 observations. This number does not, however, indicate the number of unique people this set represents, since some participated in more than one study. In particular, Studies 2, 4, and 5 were each subsets of Study 1, although not all of the people who were monitored in the later studies had been monitored in

Study 1. In some cases, a member of the same household was used, if the original person was not available. If neither of these could participate, then a new person in a new household was selected as a replacement. To create a combined data set that did not overrepresent individuals who were sampled more than once, we used the average of the measurements for those participants who were measured more than once. This procedure resulted in the following final number of observations for the combined data, depending on the target compound: 388 for benzene, 392 for styrene, 303 for ethylbenzene, 397 for m–xylene, and 397 for o,p-xylene.

We investigated the relationship between two variables using correlation analysis, which provides a quantitative measure of the degree to which one or more variables can predict the value of another variable. It measures the strength of linear relationships only and cannot determine whether the relationship is causal; it can only indicate whether the degree of common variation is statistically significant or not.

Two correlation indices are commonly computed to quantify the relationship between variables: the Pearson and the Spearman rank correlation coefficients. The Pearson coefficient indicates the ratio of the explained variation in the values of the variables to the total variation in the values: if the absolute value of the coefficient equals one, the explained variation equals the total variation, indicating a perfect association; conversely, if the coefficient equals zero, the predictive variables explain none of the variation. A strong correlation is indicated when the coefficient is determined to be significantly different from zero using hypothesis testing (Bevington and Robinson, 1992); a value of the Pearson coefficient whose magnitude exceeds 0.5 can also indicate good correlation (Burmaster and Anderson, 1994). The sign of the coefficient tells whether one variable increases with an increase in the other variable (positive) or whether one variable decreases with an increase in the other (negative).

The Spearman rank coefficient uses the ranks of the values of the variables instead of the values as measured on the continuous scale. It is thought that for certain types of data, such as those with widely varying values, the Spearman rank coefficient is a better representation of the degree of association between two variables than the Pearson coefficient (McCuen, 1985; Wallace, 1987). Because the ETS exposure data exhibit large variability, and although we computed both unweighted Pearson and Spearman rank correlation coefficients, we believe that the Spearman rank coefficient is a better indicator for exploring correlations within the CED. For more information on correlation coefficients, see McCuen (1985).

We also used frequency tables to explore relationships between continuous exposure variables and those variables that were either categorical (sex: male or female) or had a range of values (age). This analysis was conducted using the combined data set. Twenty-four-hour personal exposures were sorted into four categories using the 25th, 50th, and 75th percentiles of

the combined data as cutoff points. These four categories were cross-tabulated with other variables of interest to produce frequency counts and percent proportions.

B.2.1 Yearly Variation (Study 1 versus Studies 4 and 5)

To better understand the temporal characteristics of exposure, we examined the correlations between exposures over pairs of years, comparing concentrations measured in Study 1, conducted in 1984, with those from Studies 4 and 5, conducted in 1987. Studies 4 and 5 are subsets of Study 1. Not all of the participants in these studies, however, had participated in Study 1: only 32 of the 51 participants in Study 4, and 26 of the 43 participants in Study 5, participated in Study 1. In this analysis, we used only concentrations measured for the same people in two different studies.

The results of our analyses, presented in Table B.8, suggest that exposures do show a reasonable level of correlation over time. To be specific, a statistically significant correlation in this context indicates that individuals exhibiting high exposures in the first study also tend to exhibit high exposures in the subsequent study, and vice versa. Four of the five compounds (styrene was the exception) had Spearman rank coefficients higher than 0.5 when comparing Studies 1 and 4, both of which were conducted during the winter. Only two of five compounds had Spearman coefficients higher than 0.5 when comparing Studies 1 and 5, which occurred in different seasons. A similar analysis of Studies 2 and 5, both of which were conducted during summer months, yielded sample sizes of between 10 and 14 people, which we considered too small to reveal correlation trends.

Yearly correlation results were inconsistent among compounds: the compounds which showed good correlation were not always the same when Studies 1 and 4 were compared, as when Studies 1 and 5 were compared. All five compounds, though, had Spearman values above 0.5, significant at the 0.05 level, in at least one of the two combinations of studies.

Figure B.1 illustrates the relationship between exposures measured for the same subjects in Studies 1 and 4. For all five compounds, there are a few elevated exposures which affect the regression, and could influence the overall exposure distributions. For styrene, one exposure measured during Study 4 was above 150 $\mu g\ m^{-3}$; when this point was removed, the Pearson correlation increased from 0.12 to 0.43.

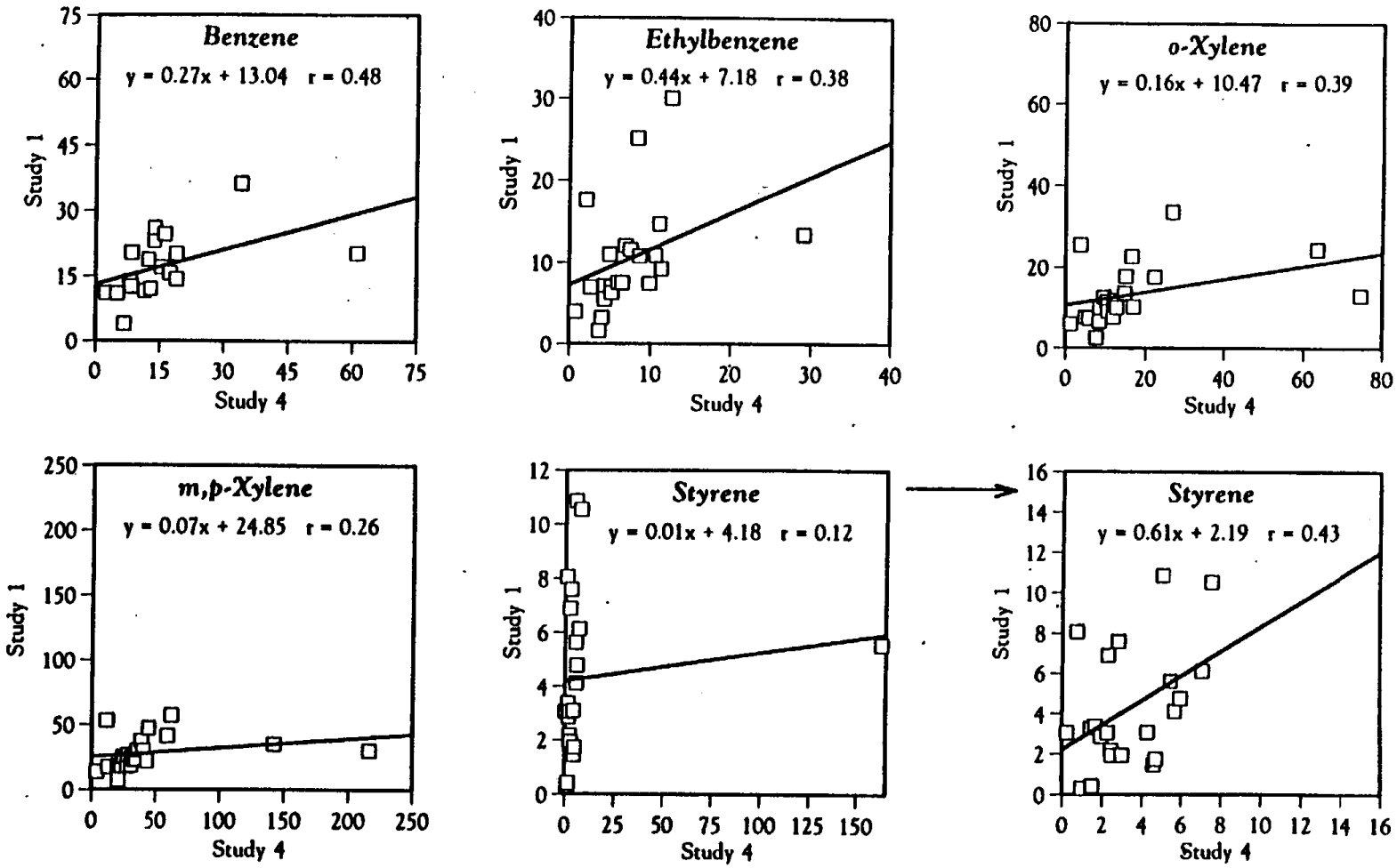

Figure B.1 Linear regression of 24-hour personal exposure concentrations (μg m^{-3}) measured for the same participant in Study 1 (1984) versus Study 4 (1987), investigating the relationship between exposures measured in different years.

Table B.9. Spearman rank correlation coefficients for comparisons of exposures to compounds for all six studies combined, within each exposure category.[1]

compound	***benzene***	***styrene***	***ethylbenzene***	***o-xylene***	***m,p-xylene***
			all		
benzene	1.00	0.65	0.82	0.84	0.85
styrene		1.00	0.73	0.70	0.70
ethylbenzene			1.00	0.94	0.95
o-xylene				1.00	0.96
m,p-xylene					1.00
			exposure category = active		
benzene	1.00	0.70	0.78	0.84	0.81
styrene		1.00	0.73	0.75	0.75
ethylbenzene			1.00	0.91	0.92
o-xylene				1.00	0.93
m,p-xylene					1.00
			exposure category = passive		
benzene	1.00	0.62	0.85	0.81	0.85
styrene		1.00	0.75	0.69	0.70
ethylbenzene			1.00	0.97	0.96
o-xylene				1.00	0.98
m,p-xylene					1.00
			exposure category = unexposed		
benzene	1.00	0.62	0.83	0.83	0.85
styrene		1.00	0.70	0.69	0.65
ethylbenzene			1.00	0.94	0.94
o-xylene				1.00	0.97
m,p-xylene					1.00

[1] All ccorrelation coefficients are significantly different from zero at $p < 0.05$.

B.2.2 Seasonal Variation (Study 1 versus 2, and Study 4 versus 5)

We compared Studies 1 and 2, and Studies 4 and 5, to investigate seasonal differences in exposures to the same compounds. These combinations of studies represent winter and summer exposures in 1984 and 1987. Hartwell, et al. (1987) reported that winter exposures from Studies 1 and 2 tend to be higher by a factor of two to three, than the corresponding summer exposures to the same compounds. The univariate statistics for 24-hour personal exposures that we computed for each study confirms this result. The mean values of a majority of compounds were two to three times higher in winter than summer, although a few compounds were higher by even greater amounts. The largest difference is seen for styrene, for which the average exposure for the active ETS exposure category in Study 4 was 17.7 $\mu g\ m^{-3}$, while in Study 5 it was 1.8 $\mu g\ m^{-3}$. Indoor air concentrations also follow this trend, as documented in Table A.1. For styrene, the average concentrations in smoking homes were 3.2 and 1.3 $\mu g\ m^{-3}$ for Studies 4 and 5 respectively.

A factor that could contribute to the higher winter exposures and indoor air concentrations is the change in residential air exchange rates between seasons. The most recent data on seasonal differences in residential air exchange rates is presented in Murray and Burmaster (1995). In Northern California, the mean rates are 0.47 and 0.68 h^{-1} for winter and summer, respectively. For Southern California, the mean rates are 0.63 and 1.57 h^{-1} for winter and summer. For a given pollutant emission rate, a lower air exchange rate tends to elevate indoor air concentrations and consequently personal exposures; higher air exchange rates tend to remove indoor air pollutants at a higher rate, reducing concentrations. In addition to seasonal variability in residential air exchange rates, there may also variability associated with a shorter time scale, such as daily fluctuations. For any individual house in which measurements were made, the air exchange rate could very well have been low during one visit, and yet higher at the next visit because the windows or outside doors were open. In §B.2.5, we explore the impact of opened windows and doors (that is, increased air exchange rates) on personal exposure levels.

We conducted correlation analyses to explore seasonal relationships between exposures to the same compound (see Table B.8). Exposures measured for the same individuals in the two different studies were considered. None of the analyses using studies from different seasons (1 vs. 2, 1 vs. 5, or 4 vs. 5) showed correlations as strong as those comparing studies in different years but in the same season (1 vs. 4). Comparisons of Studies 1 and 5 and Studies 4 and 5 show moderate correlations, suggesting that there is some consistency in exposures across seasons. The comparison of Studies 1 and 2, however, does not support this finding, with only one compound showing a significant Spearman coefficient.

Pellizzari et al. (1987) also reported no strong correlation between seasons in their analysis of Studies 1 and 2. In their analysis, the only compound showing any significant correlation

(Spearman) between seasons for 12-hour daytime personal exposures was benzene. For 12-hour nighttime personal exposures, the correlation was significant only for ethylbenzene and o-xylene. Our conclusion, then, is that exposures from the same season in different years are more similar than exposures within the same year, across seasons.

We also performed correlation analyses within each ETS exposure category to look for yearly and seasonal trends, but the limited sample size of the studies forced us to question the value of the results. Using only the subjects monitored in two studies gave sample sizes between 20 and 50 people. Subdividing these further into the three exposure categories left us with sample sizes as small as five people. Consequently, the results of these analyses within exposure categories are not presented here.

B.2.3 Variation among Compounds within a Study

The final correlation analysis we performed searched for trends in the exposures to different compounds within the same study. This analysis was conducted for each study, for each smoking category, and for the combined data set. The results for each of these variations showed strong correlations for all of the five compounds of interest.

In general, the strongest correlations appeared among three compounds: ethylbenzene, o-xylene, and m,p-xylene. In each of the studies[1], and in each exposure category, the Spearman coefficient for each pair of these compounds was 0.9 or higher, with a p-value < 0.0001, a very strong correlation. Wallace (1987) also reported that concentrations of ethylbenzene, o-xylene, and m,p-xylene were highly correlated in personal air in his analysis of the data from New Jersey and California TEAM studies. Both the results of Wallace and our results strongly suggest similar sources for these compounds.

Most studies also showed good correlations between other pairs of compounds. The weakest correlation was between benzene and styrene, with only three of the six studies showing a Spearman coefficient above 0.5. The one study which did not show strong correlations was Study 5: seven of the ten combinations of compounds had Spearman values below 0.5. (The exceptions were the three pairings involving the xylenes and ethylbenzene.) It is unclear why this one study showed such different results from the other five, all of which had no more than one Spearman value below 0.5.

Table B.9 presents the correlation results for the combined data set, within each smoking category. Among these four analyses, the lowest Spearman values were for benzene and styrene (lowest = 0.62). Again, the highest coefficients were for ethylbenzene, o-xylene, and m,p-xylene.

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[1]Except study 6, which did not measure ethylbenzene concentrations.

All correlations were significant at $p < 0.05$. These results support the hypothesis that common factors control exposure to these compounds.

B.2.4 Smoking Characteristics versus Exposure

We created frequency tables to see if higher exposures consistently occurred in the active and passive exposure subpopulations compared to the unexposed subpopulation. Personal exposures (24-hour) were sorted into four quartile categories (< 25th, 25–50th, 50–75th, and > 75th percentile). We conducted this analysis with the combined data set for benzene, ethylbenzene, styrene, o-xylene, and m,p-xylene. We also performed this analysis for each study separately, but this resulted in some bins containing too few observations to reflect trends in the data.

For all compounds, there was a strong relationship between the personal exposure level and the exposure category. For each compound, the participants with active status were almost twice as likely to appear in the higher quartiles than in the lower ones. Persons in the passive category showed no clear trend for benzene exposure, and had only slightly higher tendency to appear in the lower quartiles than active smokers. The unexposed population appeared in higher numbers in the lowest quartiles. Details for benzene are shown in Figure B.2. Fifty percent of the lowest quartile comprises those unexposed to ETS; this number decreases to 37% for the highest quartile. Twenty-three percent of the lowest quartile exposures are for active smokers; this number increases to 39% for the highest exposures. In summary, this analysis suggests that participants in the active exposure category show up more often in the highest exposure bins, with passive and those unexposed showing up in correspondingly lower bins.

We investigated other smoking characteristics, such as the average hours per day enclosed with smokers either at home or at work. Generally, as the average hours enclosed with a smoker increased, the percentage of higher exposures increased. This trend was apparent for both home and work, and for most compounds. Figure B.3 illustrates the relationship between the number of cigarettes smoked per day in an average week and benzene exposures, for those who reported smoking cigarettes in the combined data set. The percentage of heavy smokers (> 30 cigarettes per day) increased from 12% of the lowest quartile to 20% of the highest quartile, and the percentage of light smokers (1–10 cigarettes per day) decreased from 32% of the lowest to 20% of the highest quartile.

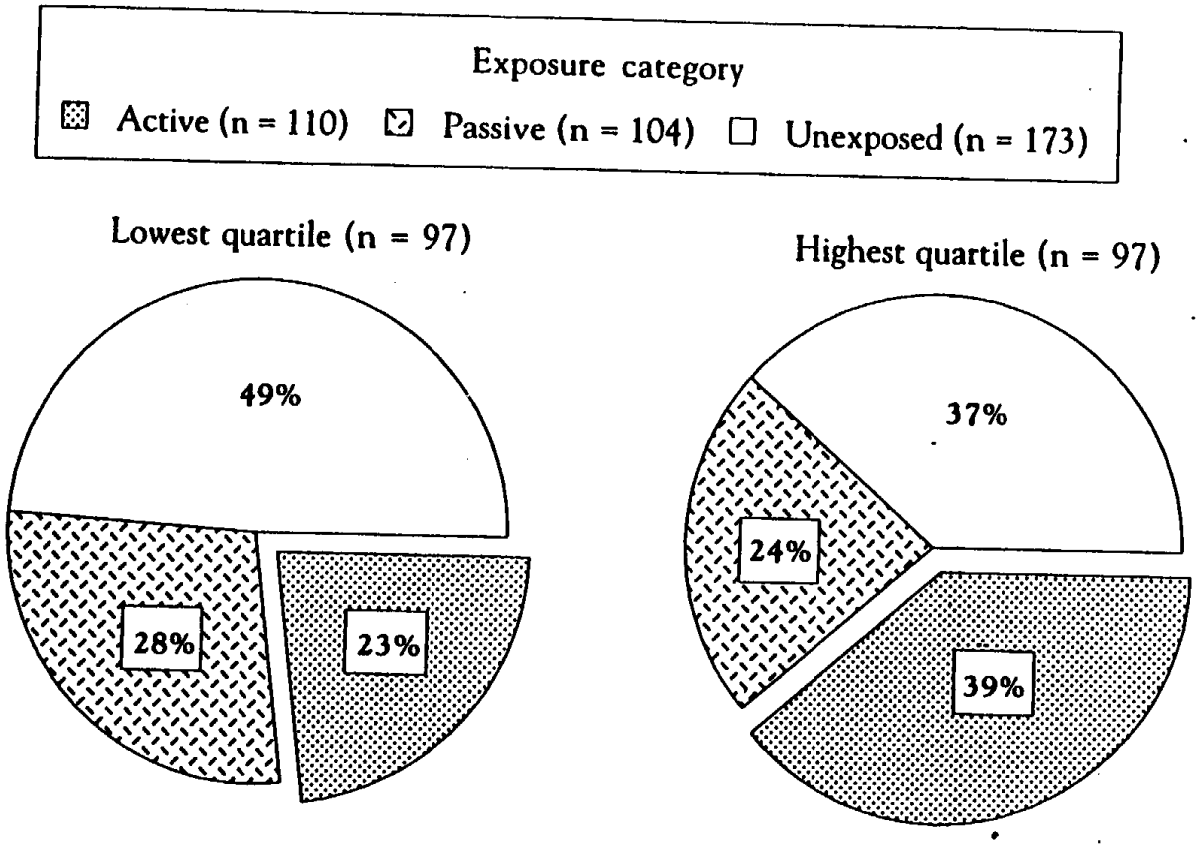

Figure B.2 Proportion of 24-hour exposures to benzene for the combined CED data set, segregated by exposure category.

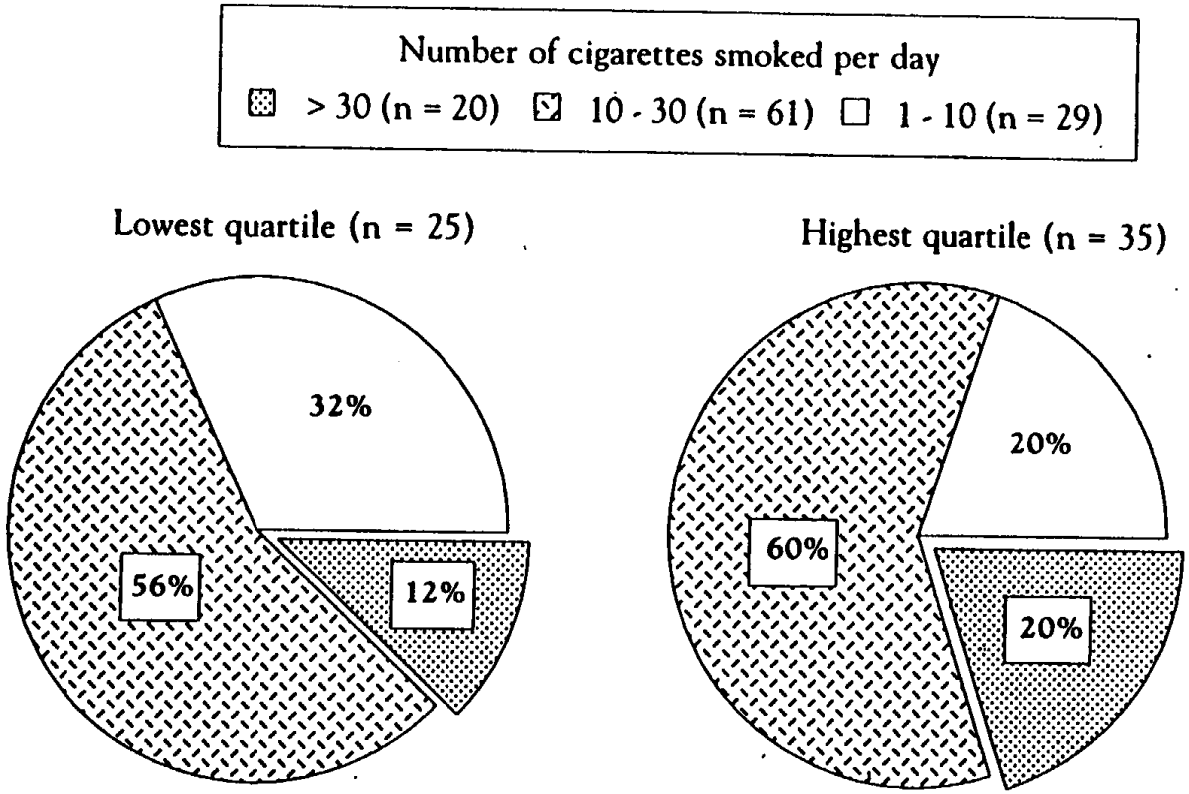

Figure B.3 Proportion of 24-hour exposures to benzene for the combined data set, segregated by number of cigarettes smoked per day.

B.2.5 Other Activities and Exposure

We produced frequency tables to investigate relationships between exposure status and a variety of household characteristics and participant activities. Some characteristics clearly showed no relationship, such as the presence of a gas furnace in the house, whether a person used cleaning solutions on the study day, or whether a person refueled their car during the study. Others, however, showed some association for one compound, but not another, or even a reverse dependence from what would be expected. For example, participants who mentioned that painting took place in the home close to the time of the monitoring period showed lowered exposures to styrene. The use of paints or solvents on the study day tended to increase exposure levels to ethylbenzene: 14% of those who reported using paints or solvents had exposures in the lowest quartile compared to 41% in the highest quartile; of those participants who did not use paints or solvents, 26% and 23% had exposures in the lowest and highest quartiles respectively.

Opening the windows or outside doors during the monitoring period showed an association with exposure for all five compounds. Figure B.4 shows that participants who reported open windows or outside doors on the study day were twice as likely to appear in the lowest quartile as in the highest quartile.[2] The increased likelihood of reduced exposures for homes with more natural ventilation supports the idea that there are indoor sources of these TACs and that increasing ventilation rates can reduce exposure.

[2]More than half of the participants (227–234 people) did not respond to this question. Of those participants who did answer, only 4–8 reported windows or doors closed on the study day. Thus the relationship between exposure level and closed windows or doors could not be analyzed due to the limited number of observations.

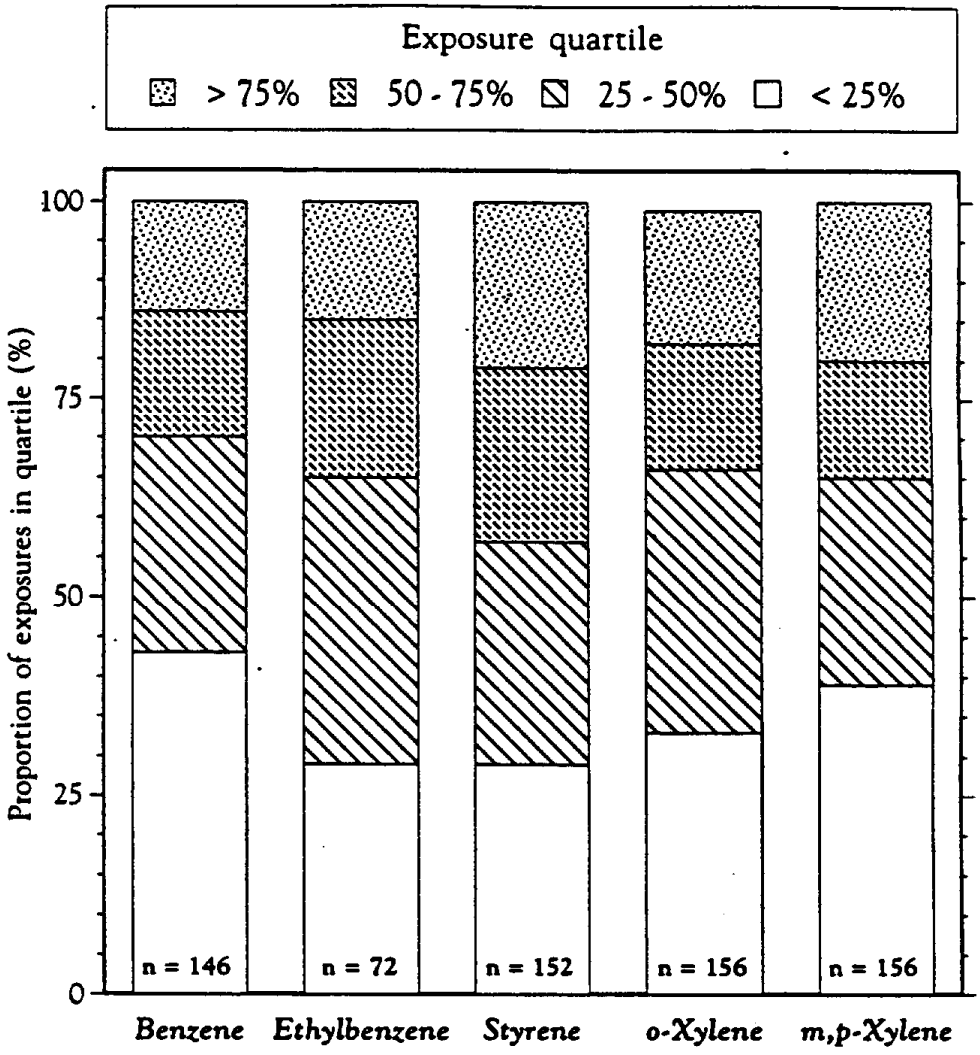

Figure B.4 Proportion of 24-hour exposures for participants who opened their doors or windows during the monitoring period.

B.2.6 Demographics and Exposure

The main demographic information included in the studies was the sex, race, and age of the participants. We used frequency tables to explore possible relationships between exposure and these characteristics. Sex showed no significant relationship with exposure to any of the measured compounds. Explorations of age versus exposure showed that children (age 14 or under) generally incurred lower exposures, while the largest number of high exposures were for ages 30–44. For example, 67% of the exposures to benzene and styrene for children (age 14 or under) were in the lower two quartiles.

The race of the participant correlated with exposure in the combined data set. For benzene, 44% of all Hispanics showed up in the highest quartile, and for styrene, 50% of the Blacks were in the highest quartile. It should be noted that the population of non-White races was significantly smaller than the White population in all the studies. For example, the combined data set contained 296 observations with measurable concentrations of styrene. Of these, 208 were White, 22 were Black, and 38 were Hispanic. Numbers of similar proportion appeared for all compounds, with Whites making up nearly 70% of the sampled population, and Blacks, Hispanics, and Asians making up approximately 10% each. Thus, while the correlation between race and exposure may appear significant, too few observations were made among non-White races to draw any firm conclusions relating to larger populations. A more detailed discussion of the demographics of the six studies appears in §4.3.2.

Appendix C: Deriving ETS-only Exposure Distribution from Personal Monitoring Data

This method was developed to estimate the distribution of 24-hour personal exposures that can be attributed to ETS for selected air toxicants, as outlined in §4.1. Figure 4.2 presents a schematic overview of the method. A Monte Carlo-based simulation was used to derive the distribution from monitoring data contained in the Californian Exposures Database (CED). Two sets of 24-hour personal exposures measurements from the CED were used: the subset of measurements from persons who did not smoke but were exposed to ETS during monitoring (passive), and the subset of measurements from persons who were not exposed (unexposed). This appendix presents the theoretical framework of the method and describes it in more detail.

C.1 Theoretical Framework

Let f(y) be the distribution of exposure levels, y, for the Californian passive subpopulation; let g(x) be the distribution of exposure levels, x, for the Californian unexposed subpopulation; and let h(z) be the distribution of exposure levels among a hypothetical Californian population exposed to ETS only. The task is to estimate h(z). If g(x) were uniformly equal to zero, that is, there was no background exposure except that caused by ETS, then h(z) = f(y). However, this is clearly not the case; exposure to the compounds of interest is also caused by sources other than ETS. Therefore, the task is to estimate both f(y) and g(x) from the monitoring data in the CED, and to use these results to further estimate h(z). The sample distributions observed from the monitoring data are directly used to estimate f(y) and g(x); these distribution estimates are denoted by $\hat{f}(y)$ and $\hat{g}(x)$. The estimated distribution of ETS-only exposures, $\hat{h}(z)$, is assumed to be lognormal; such a distribution can be uniquely described by two parameters: the geometric mean (GM), denoted GM_{ETS}, and the geometric standard deviation (GSD), denoted GSD_{ETS}.

C.2 Description of Procedure

The iterative procedure used to estimate $\hat{h}(z)$ begins with an initial guess of GM_{ETS} and GSD_{ETS}; these are the parameters for a first postulated distribution, $h_1(z)$. To determine whether $h_1(z)$ is a good estimate of $\hat{h}(z)$, $h_1(z)$ is used to construct a hypothetical distribution, $f_1(y^*)$, which is then compared to $\hat{f}(y)$. The distribution $f_1(y^*)$ represents what the Californian population might be exposed to if they were somehow separately exposed first to ETS and then to all other sources besides ETS; it can be thought of as a *simulated* $\hat{f}(y)$. In §4, this distribution is referred to as "*ETS-only + unexposed.*"

To construct $f_1(y^*)$, a z value is randomly selected from $h_1(z)$, which can be thought of as a measurement of the exposure to an air toxicant after elimination of all sources except ETS. An x value is also randomly selected from $\hat{g}(x)$, which can be thought of as a measurement of exposure due to all sources other than ETS. Next, these two randomly sampled points are added together to obtain a y^* value. This process is repeated until a sufficient number of y^* values are collected to estimate the hypothetical distribution $f_1(y^*)$. (In preliminary analyses, we found that 10,000 such iterations provided stable parameter estimates for $f_1(y^*)$; using fewer than 10,000 iterations resulted in parameter estimates that changed slightly depending on the number of iterations.)

The distribution $f_1(y^*)$ is compared to $\hat{f}(y)$ using the Kolmogorov-Smirnov statistic, D_{KS}. The objective of this comparison is to determine whether the hypothesized $h_1(z)$ distribution is a best estimate of h(z). The statistic D_{KS} is generally used to determine whether two samples that are drawn independently of each other are from the same population, or whether they come from distributions that have different cumulative distribution functions (McCuen, 1985; Sprent, 1989). The form of the difference in the distributions is not specified: they might have the same mean but different standard deviations; one might be skewed and one might be symmetric; and so on. Calculating the statistic is straightforward: sample cumulative distribution functions are compared. The parameter D_{KS} is defined as the largest absolute deviation in the cumulative frequencies:

$$D_{KS} = \underset{i=1}{\overset{n}{MAX}} |F_i - S_i| \qquad (C.1)$$

where F_i and S_i are the observed cumulative frequencies of both sample distributions and n is the number of items in the distribution.

The parameter D_{KS} is a function of the distribution variables GM_{ETS} and GSD_{ETS}. A grid-search method is used to iteratively determine the optimum GM_{ETS} and GSD_{ETS} by minimizing D_{KS} with respect to each of the parameters separately (Bevington and Robinson, 1992). The technique involves making an incremental change in the parameters of $h_1(z)$ to form a new distribution $h_2(z)$. The distribution $f_2(y^*)$ is then constructed, following the approach described above for $f_1(y^*)$, and a new value of D_{KS} is calculated. This procedure is methodically repeated k times until the minimum D_{KS} value is obtained. The $h_k(z)$ corresponding to this last iteration is the desired estimate of the ETS-only exposure distribution, $\hat{h}(z)$. The parameters of $h_k(z)$ are the best estimates of the lognormal distribution of exposures resulting from environmental tobacco smoke: GM_{ETS} and GSD_{ETS}.

Appendix D: Derivation of CMR Model Equations

Consider an exposure episode that begins at time t_1 and ends at time t_2. The exposure is estimated as the product of the exposure period ($t_2 - t_1$) multiplied by the time-average concentration, C_{avg}, defined by

$$C_{avg} = \frac{1}{t_2 - t_1} \int_{t_1}^{t_2} C(t)\, dt \qquad (D.1)$$

The dependence of the time-averaged concentration on the governing variables is derived from the following equation (§5.1.1.1):

$$\frac{d(CV)}{dt} = E - Q\,C \qquad (D.2)$$

First, we multiply both sides of the equation by dt and integrate over the period of exposure to obtain

$$V\,[C(t_2) - C(t_1)] = \int_{t_1}^{t_2} E(t)\, dt - \int_{t_1}^{t_2} Q(t)\, C(t)\, dt \qquad (D.3)$$

From left to right, the three terms in this equation represent (i) net accumulation of environmental tobacco smoke in the indoor environment over the course of the exposure period, (ii) total emissions from all cigarettes smoked, and (iii) total removal of tobacco smoke from the building by ventilation. Our model assumes that the accumulation term is negligible in comparison to the two terms on the right-hand side. This assumption introduces uncertainty, especially for short exposure periods, but probably not significant bias. With this assumption, equation (D.3) simplifies to this form

$$\int_{t_1}^{t_2} Q(t)\, C(t)\, dt = \int_{t_1}^{t_2} E(t)\, dt \qquad (D.4)$$

One more key assumption must be made to complete the model. Both the ventilation rate and the ETS concentration may vary independently with time. We assume that ventilation and ETS concentrations are uncorrelated. Strictly, this would be true only if behaviors that modify ventilation (for example, opening a window) are not dependent on ETS concentrations. As demonstrated below, by making this assumption, the left-hand side of equation (D.4) simplifies to

the product of three terms: the time-averaged concentration, the time-averaged ventilation rate, and the exposure period.

The left-hand side of equation (D.4) is simplified as follows. We decompose the time-varying parameters C(t) and Q(t) as sums of an invariant time-average and a fluctuating component:

$$C(t) = C_{avg} + C'(t) \tag{D.5}$$

$$Q(t) = Q_{avg} + Q'(t) \tag{D.6}$$

The fluctuating components, C' and Q', have the characteristic that their time-average is zero. Substituting for the left-hand side of equation (D.4) leads to this expression:

$$\int_{t_1}^{t_2} [Q(t)\, C(t)]\, dt = \int_{t_1}^{t_2} [Q_{avg}\, C_{avg}]\, dt + \int_{t_1}^{t_2} [Q_{avg}\, C'(t)]\, dt + \int_{t_1}^{t_2} [Q'(t)\, C_{avg}]\, dt + \int_{t_1}^{t_2} [Q'(t)\, C'(t)]\, dt \tag{D.7}$$

The right-hand side of equation (D.7) can be simplified. The parameters Q_{avg} and C_{avg} are time invariant and so can be taken outside the integrals. The second and third terms vanish: the time-integral of the fluctuating component is zero because that component is defined in such a way as to have zero mean. Thus, equation (D.7) simplifies to

$$\int_{t_1}^{t_2} [Q(t)\, C(t)]\, dt = Q_{avg}\, C_{avg}\, [t_2 - t_1] + \int_{t_1}^{t_2} [Q'(t)\, C'(t)]\, dt \tag{D.8}$$

The statement that ventilation and concentration are uncorrelated simply means that the second term on the right-hand side of equation (D.8) vanishes. The left-hand side of equation (D.4) is finally simplified to:

$$\int_{t_1}^{t_2} [Q(t)\, C(t)]\, dt = Q_{avg}\, C_{avg}\, [t_2 - t_1] \tag{D.9}$$

We now return to simplifying the right-hand side of equation (D.4). We determine the emission rate, E ($\mu g\ h^{-1}$), of species from tobacco smoke into indoor air as the product of three terms:

$$E = n\, N\, e \tag{D.10}$$

where n is the number of smokers in the indoor environment, N is the rate at which cigarettes are smoked (cig $smoker^{-1}\ h^{-1}$), and e is the emission factor for the air toxicant in ETS ($\mu g\ cig^{-1}$). We

approximate the emissions rate, E, as constant for the waking hours, and so the time integral of E is given by:

$$\int_{t_1}^{t_2} E\,dt = n\,N\,e\,[t_2 - t_1] \qquad \text{(D.11)}$$

Substituting equations (D.9) and (D.11) into equation (D.4) leads to this expression:

$$C_{avg} = \frac{n\,N\,e}{Q_{avg}} \qquad \text{(D.12)}$$

The ventilation rate is obtained as the product of two terms

$$Q_{avg} = \lambda_{avg}\,V \qquad \text{(D.13)}$$

where λ_{avg} is the time-averaged air-exchange rate and V is the building volume. And so, the model equation we applied becomes

$$C_{avg} = \frac{n\,N\,e}{\lambda_{avg}\,V} \qquad \text{(D.14)}$$

Appendix E: Testing the Monte-Carlo Model for Predicting Exposure

Part of the process of determining the final form of the model applied in Phases II and III of this project involved performing tests to see how its configuration would affect any output we generated. In the first of these, we looked at the effect of changing the initial seeds used in the random number generators of the model. The second test was a convergence test, where we determined the minimum number of iterations required to converge on a mean exposure. The results from these tests are described in the next two sections.

E.1 Seed Tests

In each iteration, our model employs a Monte-Carlo sampling method to determine the values of several parameters. This method entails randomly sampling from a known distribution of values in which the probability of drawing a given value is determined by the parameters of the distribution. Computationally, we used a random number generator function built into the SAS software that required the use of an initial seed value. We used different initial seeds for each parameter to avoid introducing invalid correlations between parameter values.

As a test, we looked at the effect that changing the seeds used in these random number generators would have on the output generated by the model. We used residential exposure with benzene concentrations calculated using the CM method as the test case. To perform the test, we ran the model 100 times, each time incrementing the value of all of the seeds. We then looked at the resulting changes in the AM, SD, GM, and GSD of the distribution of exposures from each run.

The results of this test showed that changing the random number generator seeds resulted in only small variations in the model output. The arithmetic mean residential exposure varied by less than a factor of two, while the maximum and minimum values of the GM and GSD differed by less than ten percent of their mean values. The standard deviation predicted by the model varied the most of these four, with a range just under one order of magnitude. The extreme values were within a factor of four of the mean SD of these runs.

These results were encouraging, since they indicate that our choice of seeds would have less impact on our results than our choice of calculation methods (CMR vs. tracer) or our choice of parameter descriptors (AM/SD or GM/GSD that defined the distribution of values for a given parameter).

E.2 Convergence Tests

We performed another type of test of our model to determine the number of iterations required to converge on a mean exposure. We did this test at three computational levels: total exposure, exposure within a single microenvironment, and exposure for a single person. In each case, the method was the same: we ran the model many times, increasing the number of iterations each time until the resulting AM differed by less than ten percent of the previous run. Because the number of iterations was limited by the number of participants in the APCR study, we sampled each person multiple times to increase our sample size.

Results of this test showed that the sample size required for model convergence differed for each computational level we tested. Total exposure converged using a sample size of 14,150 (sampling each person 25 times), whereas microenvironmental exposures converged using a sample size of > 20,000. Thus we chose for our model a sample size of 22,000 which requires sampling each nonsmoking adult APCR participant 40 times.